A PROSPECTIVE ANALYTICAL STUDY COMPARING THE EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX, APACHE-II AND P- POSSUM IN PREDICTING THE MORTALITY OF PATIENTS WITH PERFORATIVE PERITONITIS.

DISSERTATION SUBMITTED FOR

BRANCH-I M.S (GENERAL SURGERY)

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THE TAMIL NADU Dr M.G.R. MEDICAL UNIVERSITY CHENNAI

CERTIFICATE

This is to certify that this dissertation titled **"A PROSPECTIVE ANALYTICAL STUDY COMPARING THE EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX, APACHE-II AND P- POSSUM IN PREDICTING THE MORTALITY OF PATIENTS WITH PERFORATIVE PERITONITIS"** submitted by **Dr JAN SUJITH. P** to the faculty of General Surgery, The Tamil Nadu Dr M.G.R Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from September 2013- September 2014.

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I have great pleasure in forwarding it to The Tamilnadu Dr. M.G.R. Medical University, Chennai.

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DECLARATION BY THE CANDIDATE

I, **Dr JAN SUJITH. P** here by solemnly declare that this dissertation entitled **"A PROSPECTIVE ANALYTICAL STUDY COMPARING THE EFFECTIVENESS OF MANNHEIM PERITONITIS INDEX, APACHE-II AND P- POSSUM IN PREDICTING THE MORTALITY OF PATIENTS WITH PERFORATIVE PERITONITIS"** is a bonafide and genuine research work carried out by me. This is submitted to The Tamil Nadu Dr M.G.R Medical University, Chennai, in partial fulfillment of the regulations for the award of MS degree (Branch I) General surgery.

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ABSTRACT

This is a prospective analytical study done on 50 patients with perforative peritonitis in Govt Rajaji Hospital, Madurai. The objective of the study was to analyse Mannheim Peritonitis Index, APACHE-II and p-POSSUM by comparing Discriminatory ability, Positive predictive value, Sharpness of prediction and Reliability of prediction. Online calculators were used to calulate risk of mortality and statistical analysis was done with SPSS Version 15 for Windows.

It was found that APACHE-II had the best positive predictive value, and discriminatory ability followed by p-POSSUM and MPI. APACHE-II also had the best positive predictive value followed by p-POSSUM and MPI. None of the scores had a very good sharpness and they were quite reliable in predicting the risk of mortality.

KEY WORDS: Mannheim Peritonitis Index, APACHE-II, p-POSSUM, Mortality, Peritonitis.

INTRODUCTION

Generalized peritonitis is a frequently lethal condition. It continues to be one of the major problems confronting physicians, surgeons and their patients throughout the world. Until the end of the last century, peritonitis was treated medically with a mortality of 90%. In 1926, Krishner showed that the mortality of peritonitis could be reduced by strict implementation of surgical principles, and the mortality rate dropped to below 50%. Since then, despite innumerable advances in surgical skills, antimicrobial agents and supportive care, the mortality of peritonitis remains high and is presently reported in various multicenter studies as varying between 13 and 43%.

The prognosis and outcome of peritonitis depend on the complex interaction of many factors, patient related, disease related and intervention related. The chronic health status is also noted to influence the outcome. Whittman demonstrated that age, duration of symptoms, white cell count, mechanisms and origin of infection are related to outcome. The outcome in most of these patients is therefore difficult to predict. Categorizing patients into different risk groups would help prognosticate the outcome, select patients for intensive care and determine operative risk, thereby helping to choose the nature of the operative procedure, e.g. damage control vs. definitive procedure. Scoring systems also help in risk stratification and in the evaluation of new diagnostic

modalities and therapeutic advances as well as in the comparison of treatment results from different clinics.

Prognostic scores are based on numerical weighting of clinical variables. Various scoring systems have been used to assess the prognosis and outcome of peritonitis. Those used include the Acute Physiological and Chronic Health Evaluation score (APACHE II), the Mannheim Peritonitis Index (MPI), the Peritonitis Index Altona (PIA), the Sepsis Score, and the Physiological and Operative Severity Score for Enumeration of Mortality and Morbidity (POSSUM). Various authors have reported APACHE to be a better system for prognostication of the outcome of patients with peritonitis, while others concluded that MPI provides a more reliable means of risk evaluation. The present study was undertaken to compare the use of three of the above scoring systems in patients with perforative peritonitis.

The MPI is based upon data from 1253 patients with peritonitis treated between 1963 and 1979 and was developed by analysis of 17 possible factors. In previous studies, patients with scores of less than 21 had a mortality rate ranging from 0-2.3% and those with MPI between 21 and 29 had a mortality rate of approximately 65%. MPI score of more than 29 had the highest mortality, up to more than 80% in some studies.

The Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM) was developed by multivariant discriminant analysis of 48 physiological and 18 operative variables by Copeland et al in 1991. Eighteen of these variables were determined to be independent factors related to patient outcome. POSSUM has since been shown to over predict mortality in low risk patient groups. The Portsmouth predictor equation (p-POSSUM) was developed to overcome this failing. Most surgeons accept that mode of presentation, physiological condition of the patient and extent of the surgical procedure performed are predictors of outcome.

The APACHE II (Acute Physiology and Chronic Health Evaluation) score integrates estimates of the severity of disease measured by 12 physiological variables with the physiological reserve estimated by age and chronic disease APACHE II is used in intensive care units to classify the severity of a disease. There are several applications for prognostic scores in peritonitis. In clinical trials they are used to define risk, to compare treatment, to define inclusion and exclusion criteria, and to measure outcome in trials that do not involve comparisons. The use of scores for the accurate and reliable prediction of mortality in individual patients with peritonitis has not yet been analysed fully.

AIMS & OBJECTIVES

1. To calculate and compare the positive predictive value of Mannheim peritonitis index, p-POSSUM and APACHE-II scores for each of the patients.

2.Compare standard cut offs for predicting mortality with cut offs obtained in the study.

3.To calculate the discriminatory power of each index by plotting Receiver Operator Characteristic curves(ROC) .

4.To determine the reliability of prediction and sharpness of prediction.

REVIEW OF LITERATURE

HISTORY

Physicians in antiquity dreaded abdominal complications. Despite the fact that peritonitis was extremely common, reports of successful surgical interventions were only anecdotal before the past century. Medicine's comprehension of the pathophysiology of the peritoneal cavity is still evolving. The history of our understanding of the process could be considered to be as recent as the current study. Despite this, the mortality rates for patients with secondary peritonitis have fallen in the last century from almost 100% to <10%.

One of the earliest references to peritoneum can be found in Edwin Smith Papyrus which was copies around 1700 years ago which is supposed to have been written around the time of Imhotep (the Egyptian patron god of medicine). Breasted who translated these works wrote in his translation. *"I felt as if I had been peering through a newly revealed window, opening upon the once impenetrable gloom enveloping man's earliest endeavors to understand the world he lived in. It was as if I had watched a hand slowly raising the curtain that covered this window, and then suddenly the hand had refused to lift, the curtain further".* The curtain may have meant peritoneum.

Since the beginning of recorded medical history, human beings have been confronted with the spectra of peritonitis. Accounts from a variety of early societies have little doubt that our ancestors recognized the value of therapeutic

drainage. In a German translation of the writings of Hippocrates appears the first through description of a patient with peritonitis. “The patient looks sick and wasted. The nose is pointed, the temple sunken, the eyes lay deep are rimmed and dull. The face expresses fear, the tongue is furrowed, the skin shiny. The patient avoids all movement and breathes shallow. The abdominal wall is rigid with muscular guarding; no bowel sounds can be heard. The pulse is quick and small. A hard, tender mass in hypochondrium is a bad prognostic sign if it involves the whole area. The presence of such a mass at the beginning of the fever indicates that death is imminent”. The above description is now known as **Hippocrates facies.** He also described septic shock as *“A protrusive nose, hollow eyes, sunken temples, cold ears that are drawn in with the lobes turned outwards, the forehead’s skin rough and tense like parchment and the whole face greenish or black or leadened”.*

In the second century A.D. Galen served as the physician to Roman citizens, gladiator and emperors. He is reported to have performed many surgeries including suturing of lacerated bowel. He wrote much about appearance of suppuration in post-operative period. In fact, Galen believed that such suppuration was critical for proper wound healing and should not be disturbed (laudable pus). Galen’s writings were revered as unshakable tenets and restrained the development of medicine and physiology for almost 1500 years. From the time of the fall of the Roman Empire until the beginning of the

16 century, medicine can be characterized as magical with strong religious overtones. The fate of surgery was sealed for centuries with Pope Innocent III religious decree of 1215 known as *"Eccelsia Abhorret de Sanguine"* , literally translated as "The Church Abhors bloodshed" . It was only at the birth of renaissance that the mysteries of the abdominal cavity began to be known. This can be attributed to the wondrous drawings of the Michelangelo, Leonardo da Vinci and Vesalius. .

Peritonitis due to perforation of acute peptic ulcer was first described by Littre in 1670. The patient was a lady of high rank, Henrietta Anne, Duchess, of Oreans and daughter of Charles I of England. John Hunter, renowned for his surgical exploits, suggested that laparotomy might be possible and even useful in the treatment of peritonitis. Hertein, in 1767, reported a cure of biliary peritonitis in dogs using irrigation of abdomen. The three developments that fostered an understanding of the peritonitis disease process included the foundation of experimental physiology by Francois Magendie and Claude Bernard, an understanding of cellular pathology as championed by Rudolph Virchow, and the advent of the germ theory by Pasteur and Koch. George Wegner first reported in 1879, a series of experiments attempting to elucidate the normal physiology of the peritoneum. The modern era of our understanding of the peritoneum was begun by John B. Murphy of Murphy Button fame.

In 1908 he wrote *"There are no stomata or stigmata in the peritoneum. The endothelial lining is everywhere, continuous"*[1] Of course, we know it is not fully true as of today. Herbert E Durham[2] analyzed fluid from peritoneal cavity and proposed a time line of cellular events, which he divided into 5 stages – (1) the stage before leukopenia, (2) the leukopenic stage, (3) the microxyphil stage, (4) the macrophage stage and (4) recovery to normal.

The experiments of Meleney[3] in the late 1926's showed that bacterial synergism existed. They showed that combinations of aerobic and anaerobic bacteria produced before sepsis than from individual strains.

REVIEW OF CURRENT LITERATURE

Definition of organ failure and evolution of scores

Some of the early systematic attempts to define the severity of surgical infection and risk of death derived from the observation that patients dying after surgical infection often followed a clinical course characterized by sequential organ failure. This has been called the "multiple organ failure syndrome" Fry and associates showed in 1980[4] that death after major operative procedures or severe trauma was usually due to infection and became more likely as the number of failed organs increased i.e. the mortality rate with no organ failure was 3%, rising to 30% - single Organ failure, 100% - > 4 organ failure.

In 1982 Knaus and others proposed a scoring system to be used for classifying patient admitted to ICU. They devised a 2 part scale. It included physiological portion, APS-34, examines abnormality among 34 possible physiological assessments (APS-34), which obtained during the first day of admission. The second part of the score is a chronic health evaluation (CH). This examines the patient's pre-admission health by reviewing the medical history for details concerning functional status, productivity and medical attention during 6 month before admission. The combination is called APACHE. This system is not specific for intra-abdominal infection. It was later modified using only 12 values the APACHE II. Pine and associates (1983)[5] confirmed the above findings. In addition, they looked at a number of other risk

factor thought to influence the development of organ failures on death and identified clinical shock at any time, malnutrition, alcoholism and age as important predictive factors. The papers by Pine and Knaus and their colleagues were the first to provide clear definition of "organ failure".

Stevens (1983)[6] recognized the need for more precision and for a greater range of potential values and devised a scoring system to represent the magnitude and severity of organ failure. He defined 7 organ systems and assigned score of 0-5 in each system. Scores were calculated by squaring the values assigned to each organ system and adding the 3 highest scores to arrive at "sepsis severity score". He based the practice of squaring the individual scores up the experimental increase in the mortality as the progressive organ system failure. Knaus and Coworkers (1985)[7] extended these observations in a report covering 5,677 ICU admissions and 2719 patients who developed organ failure.

Teichmann and associates (1986)[8] in a report concerning scheduled reoperation for diffuse peritonitis, referred to Peritonitis Index Altermheir (PIA). This used age, extent of infection, malignancy, CVS risks and leukopenia to stratify patients.

Wacha and Coworkers (1987)[9] developed a separate peritonitis index, the Mannheim Peritonitis Index (MPI) with incorporated information regarding age, gender, organ failure, cancer, duration of peritonitis, involvement Of colon,

extent of spread within the peritoneum and the character of peritoneal fluid to define risk. Scores range from 0 -47. Gen. Health, concurrent illness, arterial hypotension at the time of admission, delay in surgery and severity of peritoneal contaminations, some of the factors contributing to the post-operative morbidity and mortality- Kohli et al in a study in 1988[10].

In 1990, Verma and others[11] in PGI, Chandigarh, compared prognostic factors in peritonitis due to trauma. They found pre-operative shock, multiple hollow visceral injury, septicemia, and location of injury (colon and duodenum were significant prognostic factors and with high mortality).

In 1992, Bartel and other did a study of utility of programmed relaparotomy in diffuse peritonitis. It concluded that eradication of source of infection during first laparotomy, S.Creatinin, Patients age and pre-existing hepatic disease influenced outcome.

In 1994, Demmel N[12] compared Apache II with MPI, they concluded that there was no significant different in prognostic value between scoring systems. Khosrovanin 1994, identified 3 important prognostic factors for high mortality – age over 70 years, admission delay in > 24 hours and pre-operative hemodynamic shock. He recommended suture of perforation and vagotomy in absence of risk factors and simple suture of perforation in presence of single factor.

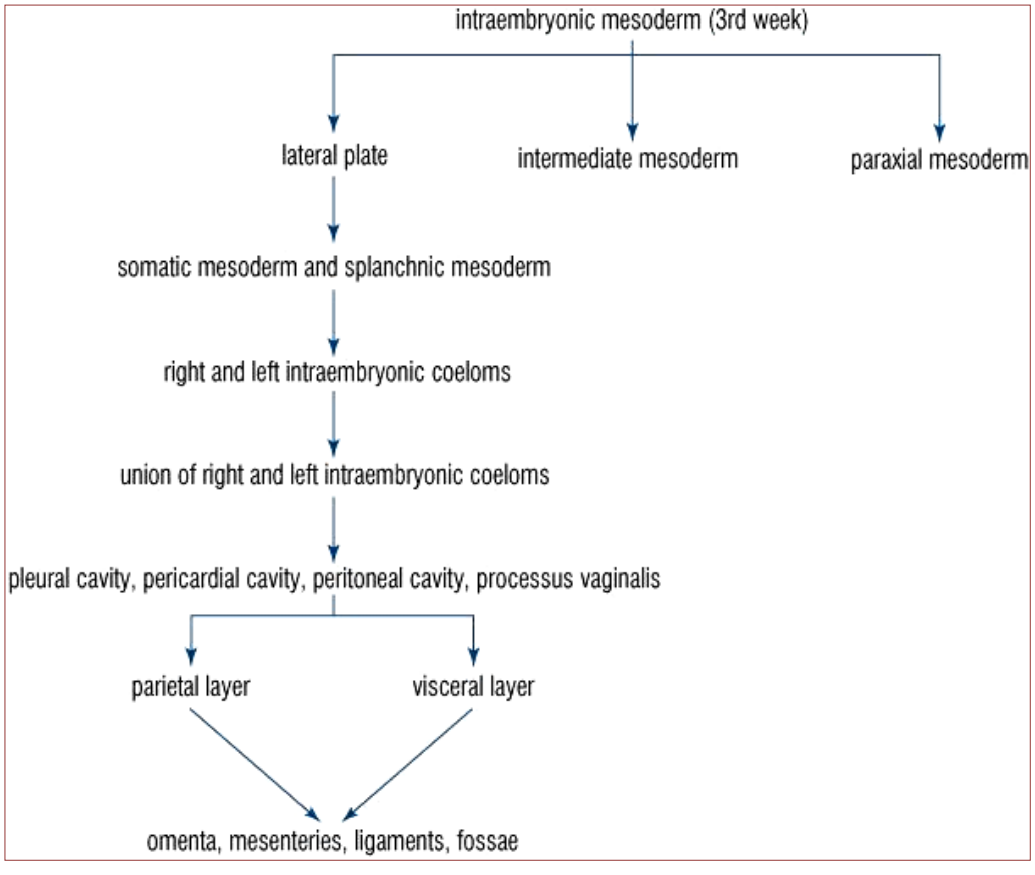
In 1994, Kriwanek S. conducted a study for prognostic factors in colonic perforation. It concluded that age over 65 years and MPI proved to be the only risk factors of significance. In 1994, Scoanes[13] and other did a study of diverse effect of delayed treatment for perforated peptic ulcer. They concluded that delayed treatment for > 12 hrs. Increased mortality especially in elderly patient confirming finding of MPI .

In 1996, a multivariate analysis on 604 patients with intra-abdominal infection were done to compare different scores systems like Apache-II, SS of Elebute and Stoner and infection on the prognosis of patients. Both Apache-II and MPI correctly graded intra-abdominal infections and were strongly and independently associated with an outcome. However, the MPI has the advantages of being easier to calculate.

SURGICAL ANATOMY OF PERITONEUM AND PERITONEAL CAVITY

Embryology of peritoneal cavity

Peritoneal cavity is derived from the two limbs of the horseshoe shaped intra-embryonic coelom, which is situated caudal to septum transversus. The two parts are at first separate, but fuse to form one cavity as result of lateral folding of embryonic disc. The attachment of mesentery of the primitive gut on the abdominal wall is initially in the midline. As a result of changes involving the rotation of the gut and as a result of some parts of the gut becoming retroperitoneal, the line of attachment of mesentery becomes complicated[14] .The peritoneal cavity therefore comes to be subdivided into number of pockets that are separated partially by folds of peritoneum.



Parietal peritoneum

It lines the inner surface of the abdominal and pelvic walls and other lower surface of the diaphragm. It is loosely attached to the walls by extra peritoneal connective tissue and can therefore be easily stripped. Because of somatic innervations it is pain sensitive.

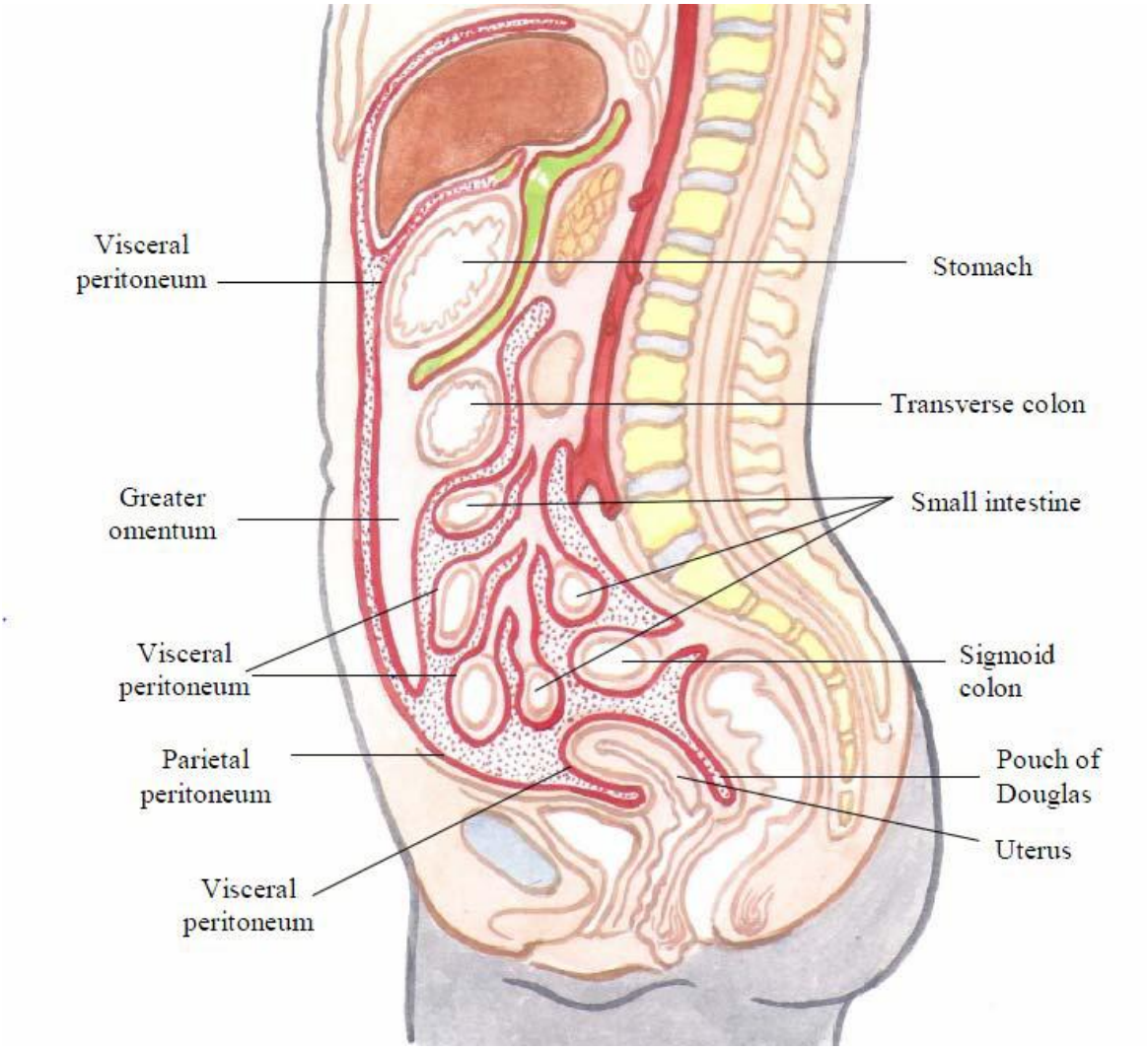


FIG 1 PERITONEUM- VISCERA AND PARIETAL LAYER

Visceral peritoneum

It lines the outer surface of the viscera, to which it is firmly adherent and cannot be stripped. Blood and nerve supply are same as those of underlying viscera. Because of the autonomic innervations it is pain insensitive[15]. Histologically, peritoneum is composed of an outer layer of fibrous tissue, which gives strength to the membrane and an inner layer of mesothelial cells which secrete a serous fluid. The peritoneal cavity is the largest cavity in the body. The surface area of its lining membrane is two square meters in adult, nearly equal to that of skin. In males, it forms a closed sac. In females, the free ends of uterine tube open into the abdominal cavity. The peritoneal cavity consists of a main region termed the Greater sac and the lesser sac (omental Bursa). The peritoneal cavity is divided into pelvic and abdominal portions. The abdominal portion is divided into supracolic and infracolic compartment by transverse colon and mesocolon. The infra colic compartment is divided into right and left by mesentery. The Right infracolic and left infracolic is divided into external and internal paracolicgutters by ascending and descending colon respectively. Supracolic compartment is below the diaphragm and above transverse colon and mesocolon. The liver, gallbladder, stomach, first part of the duodenum and spleen lie in this space. The liver and its ligaments break this space into important sub phrenic spaces

Subphrenic spaces

There are seven subphrenic spaces, four intraperitoneal spaces and three extra peritoneal spaces. It is divided into right and left by falciform ligament. The intraperitoneal spaces are:

1. Right anterior (superior) (subphrenic)

2. Right posterior (inferior) (subhepatic)

3. Left anterior (superior) (subphrenic)

4. Left posterior (inferior) (subphrenic)[16]

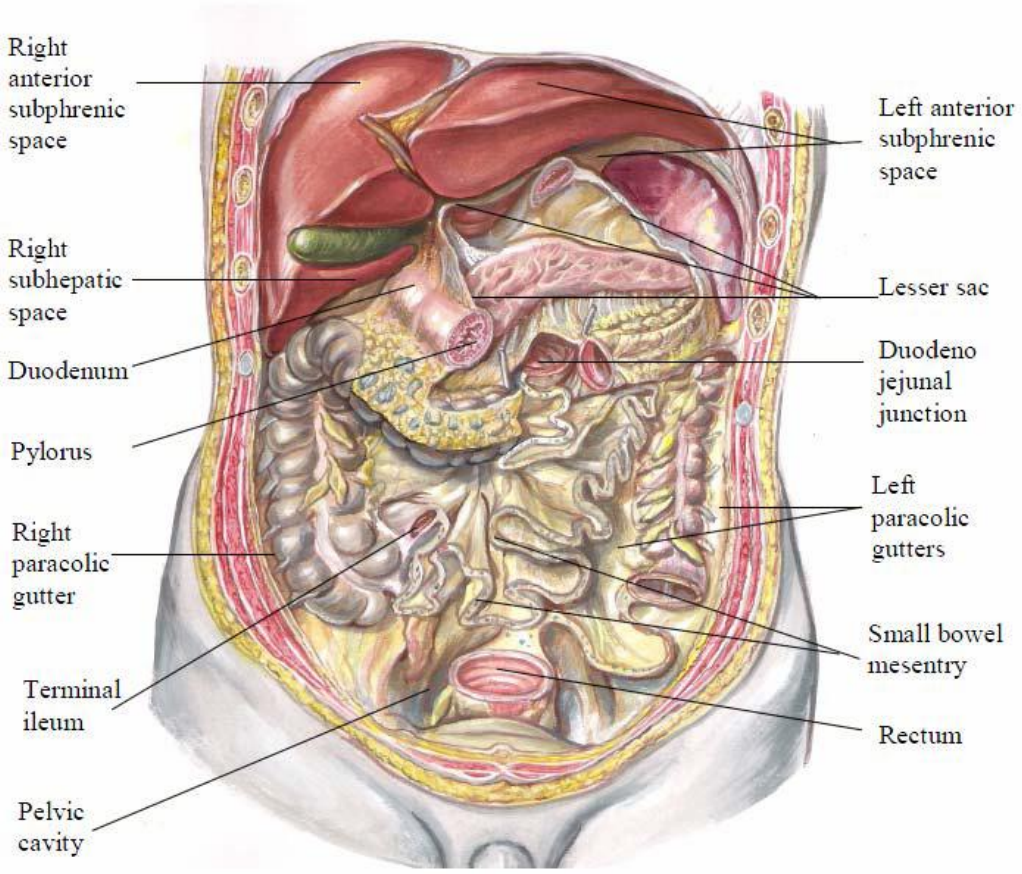


FIG 2 PERITONEUM CAVITY AND SPACES

There are three extra peritoneal spaces, which are

- Right and left extra peritoneal space also known as perinephric spaces.
- Midline extra peritoneal also for the bare area of liver.

1. Right anterior (superior) intraperitoneal space (Right subphrenic space)

It lies between the right lobe of liver and the diaphragm. It is limited posteriorly by the anterior layer of the coronary and the right triangular ligaments and to the left by falciform ligament. Common causes of collection here are perforating acute cholecystitis, a perforated duodenal ulcer, a duodenal stump blow out following gastrectomy and appendicitis.

2. Right inferior (posterior) intraperitoneal space (Right sub hepatic space)

It is also called Morrison's or hepatorenal pouch. It is bounded on the right by the right lobe of the liver and the diaphragm. To the left is situated the foramen of Winslow and below this lies the duodenum. In front are the liver and the gallbladder and behind, the upper part of the right kidney and diaphragm. The space is bounded above by the liver and below by the transverse colon and hepatic flexure. It is the deepest space and the commonest site of subphrenic abscess, which usually arises from appendicitis, cholecystitis, a perforated duodenal ulcer, or following upper abdominal surgery.

3. Left anterior (superior) intraperitoneal space (subphrenic space)

It is bounded above by the diaphragm and behind by the left triangular ligament and the left lobe of the liver, the gastrohepatic omentum and anterior surface of the stomach. To the right is the falciform and to the left the spleen, gastrosplenic omentum and diaphragm. The common cause of an abscess here is operation on the stomach the tail of pancreas, the spleen or the splenic flexure of the colon.

4. Left inferior (posterior) intraperitoneal (left sub hepatic space)

It is another name for the lesser sac. The commonest cause of infection here is complicated acute pancreatitis. In practice a perforated gastric ulcer rarely causes a collect ion here because the peritoneal space is obliterated by adhesions.

5.**Extraperitoneal spaces**

The right and left extraperitoneal space is the site for perinephric abscess. Midline extra peritoneal space is another name for the bare area of the liver. This area may develop an abscess in amoebic hepatitis and pyogenic liver abscess. It can cause generalized peritonitis following rupture.

PHYSIOLOGY OF THE PERITONEUM

Mesothelial cells are of two types- cuboidal and flattened. Cuboidal cells have stoma in between them, which is increased in peritonitis. Beneath the epithelium is a collagen layer that forms the basement membrane and still beneath it is a complex connective tissue composed of mast cells, fibroblasts, eosinophils, lymphocytes etc[15]. Mesothelium secretes around 50- 150 ml of peritoneal fluid per day and its composition is similar to plasma[17.] The peritoneal fluid has around 3 gm/dl which is less than that of plasma. Mesothelium and subdiaphragmatic lymphatics absorb fluid.

Mesothelial cells also absorb solute by process of endocytosis. This bi-directional movement of fluids across peritoneal membranes has been used in peritoneal dialysis. Two primary forces govern the movements of fluids within the peritoneal cavity.

(a) Gravity

(b) Negative pressure created beneath the diaphragm with each normal respiratory cycle.

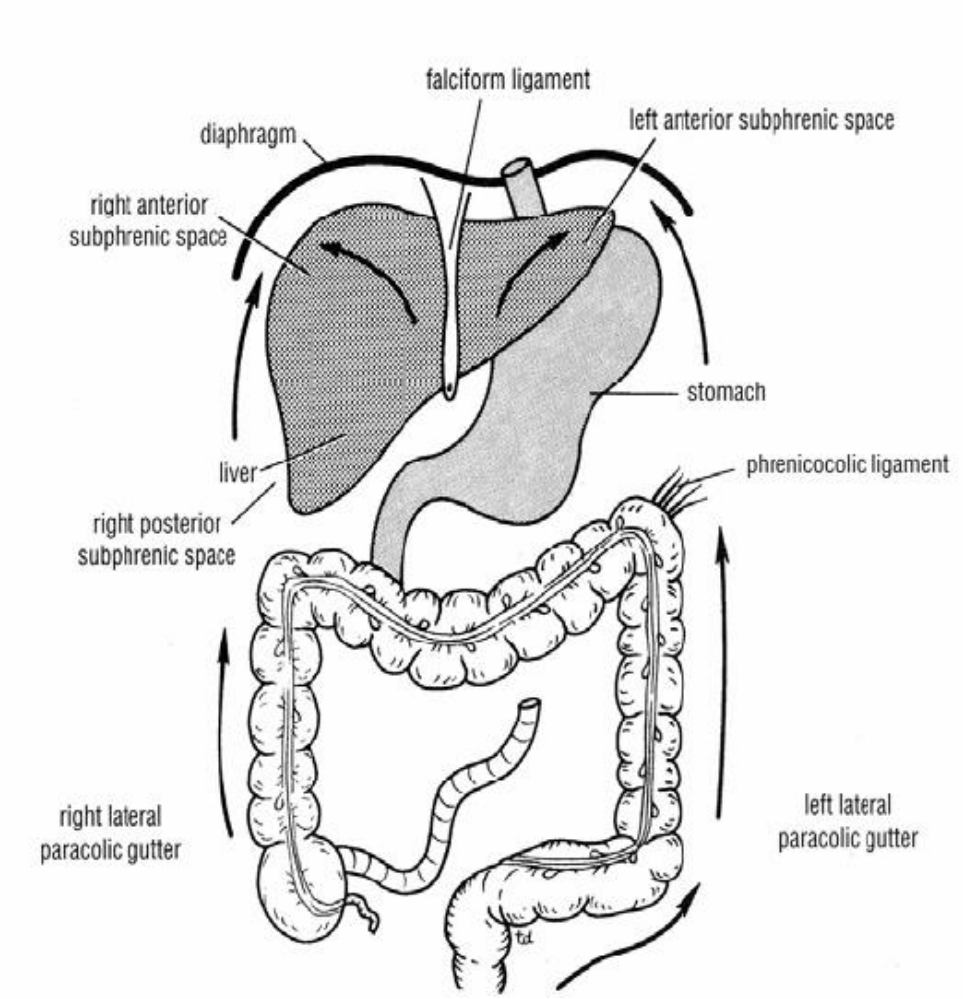


FIG: 3. NORMAL DIRECTION OF FLOW OF PERITONEAL FLUID

PERITONEAL RESPONSE TO INJURY

Any inflammatory event in the peritoneal cavity results in the peritoneal irritation with loss of regional mesothelial cells. A large peritoneal defect heals in the same amount of time as a small defect. It has been shown that after three days of peritoneal injury connective tissue cells resembling new mesothelium cover wound surface. At day five, new surface layer closely resembles adjacent normal epithelium. On day eight mesothelium regeneration is complete. The

exact origin of cells responsible for mesothelial regeneration remains unknown. It is postulated, the regeneration mechanisms include Submesothelial cells producing new mesothelial cells. Surviving or floating mesothelial cells or those attached to wound edges migrating into the wound.

Peritoneal fluid monocytes and macrophages may also be differentiating into mesothelial cells[15]. Normal peritoneal wound heals without adhesion formation. Adhesion develops in response to factors others than simple peritoneal wounding. Local tissue hypoxia or ischemia appears to be the most important factor in adhesion formation apart from mechanical sub peritoneal surface injury, intra-abdominal infections, and contamination of peritoneal cavity by foreign material. Deposition of fibrin following peritonitis is essential for adhesion formation.

It has been shown that fibrinolytic activity is absent in healing wound until mesothelial cells are found. Fibrinolytic activity is minimal at three days in view of few mesothelial cells but complete at the end of eighth day, when mesothelial regeneration is complete. Therefore with intact mesothelial surface and adequate fibrinolysins, early fibrinous adhesions disappear. Formation of adhesion is both a protective response, helping to localize infection and an adoptive response to wound healing by carrying additional blood supply.

PATHOPHYSIOLOGY OF PERITONITIS

Generalized or local inflammation of peritoneum is designated as peritonitis. Each and every case of peritonitis of whatever cause, initiates a sequence of responses involving the peritoneal membrane, the bowel, and the body fluid compartments, which then produce secondary endocrine cardiac, respiratory, renal, and metabolic responses.

PRIMARY RESPONSES IN PERITONITIS

Membrane inflammation

Peritoneum reacts to injury by hyperemia and transudation. Edema and vascular congestion occurs in the sub peritoneal layer immediately external to peritoneal membrane. Absorption across inflamed peritoneum in early cases is increased and decreases with chronicity. Absorption of macromolecules appears to be more affected than small molecule absorption. Transudation of fluid with low protein content from the extracellularly interstitial compartment into abdomen is accompanied by diapedesis of polymorphonuclear leucocytes.

During the early vascular and transudative phase of engorgement, the peritoneum acts as a two way street such that toxins and other materials that may be present in the peritoneal cavity are readily absorbed, enter the lymphatic and blood stream and may lead to systemic symptoms[15]. Transudation of interstitial fluid into the peritoneal cavity across the inflamed peritoneum is

shortly followed by exudation of protein rich fluid. The fluid exudates contains large amounts of fibrin and other plasma proteins in concentration sufficient to bring about clotting later, that results in agglutination of loops of bowel, other viscera and the parities in the area of peritoneal inflammation. There is increased synthesis of lipoproteins and proteolysis.

Concentration of uronic acid increases reflecting the exudation of plasma proteins in the early stages of peritonitis and in later stages increased synthesis of glycosaminoglycans due to activation of fibroblasts and mesothelial cells. Changes in non-collagen and collagen protein synthesis are two events that occur in inflamed peritoneum during peritonitis. In early peritonitis non-collagen protein synthesis are increased and vice versa in later stages owing to increased protein synthesis in total. The RNA: DNA ratio, an index of protein synthesizing capability of tissues, increases during the first week of peritonitis.

Bowel response

Initially, response of bowel to peritoneal irritation is transient hypermobility. After a short interval, motility becomes depressed and nearly complete adynamic ileus soon follows. Bowel distension with air and fluid accumulation occurs finally.

Hypovolaemia

Peritoneum reacts to injury by hyperemia and transudation of plasma like fluid from the extracellular, intracellular, and interstitial compartments into the peritoneal space. The loose connective tissue beneath the mesothelium of the viscera, mesentery and parieties trap extra cellular fluid as edema. The atonic bowel also accumulates the fluid derived from extra cellular space. This translocation of water, electrolytes, and proteins into a sequestered "THIRD SPACE" functionally removes this volume temporarily from the body economy. The rate of functional extracellular fluid loss is proportional to the surface area of peritoneum involved in the inflammatory process. With extensive peritonitis, translocation of 4-6 liters or more in 24 hours is not uncommon.

SECONDARY RESPONSES IN PERTIONITIS

Endocrine response

There is almost an immediate adrenal medullar response, with out - pouring of epinephrine and nor-epinephrine producing systemic vasoconstriction, tachycardia and sweating. There is increased secretion of cortical hormones during the first two or three days following peritoneal injury. Secretion of aldosterone and ADH is also increased in response to hypovolemia resulting in increased water and sodium conservation. Water retention may be greater than sodium retention resulting in dilutional hyponatremia.

Cardiac response

The effects of peritonitis and cardiac function are a reflection, both of decrease in ECF volume and progress in acidosis. Volume deficit results in decreased venous return and diminished cardiac output. Heart rate increases in an attempt to increase cardiac output, but compensation is usually incomplete. Progressive acidosis brings about secondary dysfunction in cardiac contractility and a further decrease in cardiac output.

Respiratory response

Abdominal distension, primarily due to adynamic ileus, coupled with restricted diaphragmatic and intercostal muscle movements because of pain, results in decrease in ventilator volume and early appearance of basilar atelectasis.

Renal response

Urine volume is diminished and renal capacity to handle an excess of solute is impaired. Hypovolemia reduces cardiac output and increased secretion of ADH aldosterone in peritonitis, all acting synergistically on the kidney. Renal blood flow is reduced and in turn the GFR and tubular urine flow. Reabsorption of water and sodium is increased often in imbalance and potassium is wasted.

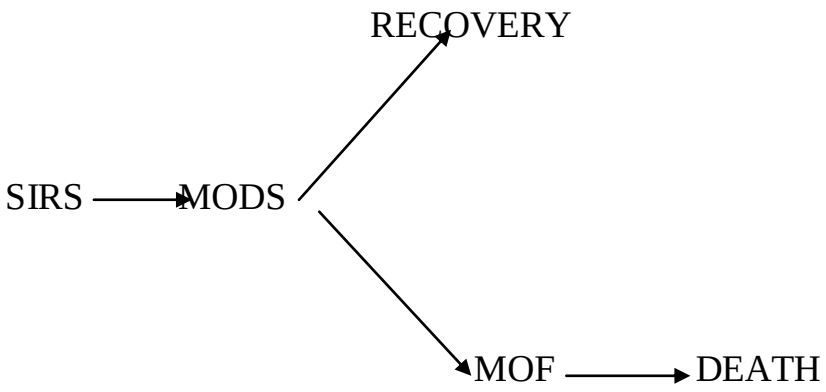
Metabolic response

The metabolic rate is generally increased with increased peripheral oxygen demand. Simultaneously the capacity of lungs and heart to deliver oxygen is reduced. Poor circulation leads to shift from aerobic to anaerobic metabolism in muscle and other peripheral tissues. As a result, anaerobic end products of carbohydrate metabolism accumulate and lactic acidosis begins to develop. Both 'D' and 'L' isomers of lactate are produced by bacterial metabolism and may be absorbed during peritonitis. Human beings can rapidly metabolize 'L' lactate, but have a relatively limited capacity to handle 'D' lactate. Protein catabolism begins early in peritonitis and progressively becomes severe. Plasma proteins are preferentially synthesized while muscle proteins are catabolized during peritonitis.

PATHOPHYSIOLOGY OF SEPSIS

Osler said *"Patients die not of their disease; they die of the physiological abnormalities of their disease,"* which is true for sepsis. Peritoneal insult will be manifested generally as Systemic Inflammatory Response Syndrome (SIRS) which if not treated aggressively will lead on to Multi Organ Dysfunction Syndrome (MODS). Bacteria can be experimentally demonstrated in thoracic duct in six minutes and in bloodstream within twelve minutes following injection of organism into peritoneal cavity Some patients succumb to death due

to Multi Organ Failure (MOF) and others recover with modern day medical care.



DEFINITIONS

1. SIRS- SYSTEMATIC INFLAMMATORY RESPONSE SYNDROME

Two or more of following clinical signs indicates SIRS

- Temp- >38 C or <36 C. 0
- Heart rate > 90/ min
- Respiratory rate > 20/ min or PaCO2 < 32 mmHg
- WBC count >12000/mm or <4000 mm or > 10% band (immature) forms.

2. SEPSIS

SIRS + documented infection.

3. SEVERE SEPSIS

SIRS + SEPSI S + Haemodynamic compromise.

4. MODS

This is a physiological derangement in which organ function is not capable of maintaining homeostasis.

Mediators of sirs

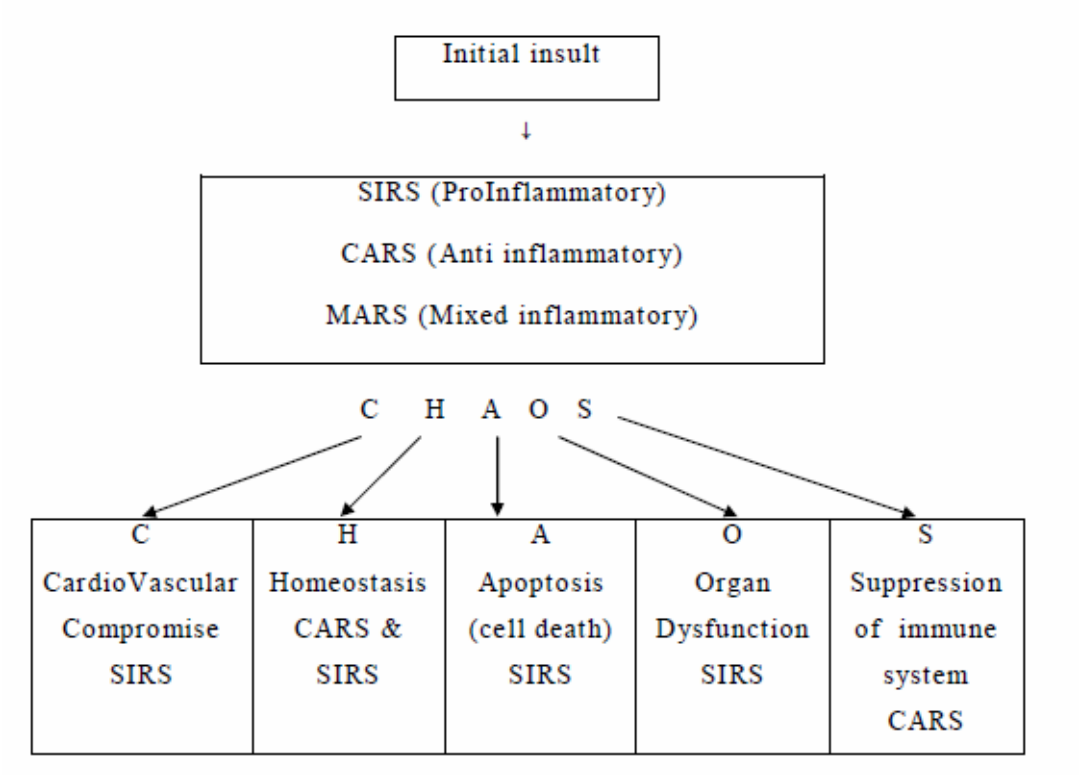
Effects of sirs are not due to one, but many mediators. The most important one is TNF (TUMOR NECROSIS FACTOR-alpha). Others are IL- 1, IL-6, Endotoxin, Endothelium and leucocytes.

Effects of sirs

There will be increased peripheral vasodilatation, microvascular permeability, microvascular clotting and leukocyte/endothelial cell activation. The metabolic and nutritional effects include fever, anorexia, cachexia etc. These effects finally lead to septic shock, DIC, ARDS and MODS.

Events in severe sepsis

After the peritoneal insult, it is postulated that initially proinflammatory (SIRS) and later anti-inflammatory responses (CARS-compensatory anti-inflammatory response syndrome) are evoked. There is also an intermediate response i.e. MARS- mixed anti- inflammatory response syndrome. The spectrum of consequences of these responses has been termed CHAOS.



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FACTORS THAT MAY FAVOUR THE DEVELOPMENT OF GENERALISED PERITONITIS

- Speed of peritoneal contaminantion is a prime factor in the spread of peritonitis.
- Stimulation of peristalsis by the ingestion of food hinders localization.
- Virulence of the infecting organism involved.
- Young children who have small omentum.
- Disruption of localized collections.
- Deficient natural resistance (immune deficiency)[15].

BACTERIOLOGY OF PERITONITIS

Peritonitis as a disease process is characteristically polymicrobial in nature

Paths of bacterial invasion of peritoneal space:

- Direct infection.
- Local extension from an inflamed organ. E.g., Appendicitis, Cholecystitis.
- Bloodstream- part of general septicemia.
- Bacteria from the alimentary canal.

The number of bacteria is low within the GIT until the distal small bowel is reached, while high concentrations are found in the colon.

The biliary and the pancreatic tract are normally devoid of bacteria, although they may be infected in the disease. Two or more organisms usually cause peritoneal infection. The commonest organisms isolated are Escherichia coli, aerobic and anaerobic streptococci, and the bacteriodes. Less frequently clostridium welchii is also found. Bacteroids are commonly found in peritonitis. These gram negative, non sporing organisms, although predominant in the lower intestine, often escape detection because they are strictly anaerobic and slow to grow on culture media unless there is adequate CO2 in the anaerobic apparatus[17].

Considerable interest has been focused on the bacterial interaction that results in a complex synergistic relationship among the pathogens of peritonitis.

Experimental studies have shown that, intraperitoneal injection of Bacteriodes fragilis alone resulted in no deaths and no lactic acidosis in rats. When *B.fragilis* is introduced into the peritoneal cavity with other aero tolerant microbes, the anaerobe becomes associated with an abscess phase of the peritoneal infection. When a large inocula B.fragilis are introduced, the mortality identified from the Endotoxin- bearing aerobic partner is accentuated. Mixed inocula of E.coli and B. fragilis show synergism in models of experimental bacteremia together.

The aerobic partners of the polymicrobial infection actually consume the oxygen of the microenvironment and generate a very low oxidation-reduction potential, which permits the non-aero tolerant anaerobes to survive.

Peritoneal infections of greatest concern are those of the distal alimentary tract, both because of the complex aerobic-anaerobic composition of bacterial pathogens and because of the very high density of bacterial contaminants.

Table 1 : Bacteria commonly encountered in peritonitis

Facultative anaerobe and Gram-negative aerobic	Obligate Anaerobes	Facultative anaerobic gram-positive aerobic
Escherichia coli	Bacteriodes fragilis	Enterococci
Klebsiella species	Bacteriodes species	Staphylococcus
Proteus species	Fusobacterium species	Streptococcus
Enterobacter species	Clostridium species	
Morganella morganii	Peptococcus species	
Aerobic gram-negative bacilli	Peptostreptococcus species	
Pseudomonas aeruginosa	Lactobacillus species	

Even in patients with nonbacterial peritonitis (e.g., intra peritoneal rupture of bladder) the peritoneum often becomes infected by transmural spread of organisms from the bowel and it is not long before a bacterial peritonitis develops.

THE ROLE OF BACTERIAL VIRULENCE

The virulence of contaminating bacteria is influenced by a number of factors. Several organisms are well recognized for their innate ability to produce intra-abdominal infection in humans. Despite the massive contamination and complexity of the microbial spectrum that occurs with caecal perforation, within 24 to 48 hours, only a few isolates are recovered in peritoneal fluid culture. This indicates that only a few pathogenic bacteria survive, to predominate infection[17]. Weinstein demonstrated that E.coli and enterococcus were the predominant organisms during the peritonitis phase[18], while B. fragilis predominated during the abscess phase. Another unique pathogenicity is the remarkable ability of encapsulated anaerobic bacteria to produce abscess formation, a characteristic attributed to the capsular polysaccharide components. The ability to adhere to the mesothelial surface may also enhance the virulence of some organisms such as the enterobacteraceae and B. fragilis. Aerobic bacteria may benefit anaerobic species by lowering the redox potential of the micro environment and producing

essential nutrients while anaerobic bacteria may provide the ability to inhibit neutrophil function and to develop antibiotic resistance by inactivation.

DIAGNOSIS OF PERITONITIS

CLINICAL FEATURES

Generalized peritonitis may present in differing ways depending on the duration of infection.

Early phase

Pain which is made worse by the movement of breathing, is almost always a predominant symptom. It is first experienced at the site of original lesion. (E.g. In case of perforated gastric ulcer pain in the epigastric region).The patient usually lies still.

Pain may be sudden or gradual in onset, varying considerably in intensity, often severe and unremitting, but at times may be no more than a dull ache. In some cases, especially in feeble and aged patients, pain may be entirely absent. Abdominal tenderness and rigidity are typically seen when inflammation involves anterior abdominal wall. Tenderness and rigidity are diminished or absent if anterior abdominal wall is unaffected as seen in pelvic peritonitis or peritonitis in lesser sac.

Patients with pelvic peritonitis complain of urinary symptoms. Infrequent bowel sounds may be heard, but ceases once paralytic ileus sets in. Pyrexia is

also present in many cases. Nausea is frequent and may be accompanied by vomiting. Fever is usually higher and more spiking in healthy young adults than infants and old aged patients. Hypothermia may occur in severely ill patients.

Vomiting may be slight at start, but as peritonitis advances, it becomes persistent. At first only the stomach contents are voided, later the fluid that is brought up is bile- stained and brownish. While finally the obstruction becomes complete, it becomes feculent. In the early stages vomiting is reflex in origin, later it becomes secondary to paralytic ileus.

A rising pulse rate and falling temperature are of gravest significance. On the other hand, a gradually rising temperature and slowly falling pulse rate suggest localization of infection is taking place. Depending on previously factors spread or localization of infection may occur. The next phase is the intermediate phase which is very deceptive.

Intermediate phase

Peritonitis may resolve, so that the pulse slows, the pain and tenderness diminish, leave a silent, soft abdomen. The condition may localize, producing one or more abscesses, with overlying swelling and tenderness.

Terminal phase

If resolution or localization has not occurred, the abdomen remains silent, and increasingly distends. Circulatory failure ensues, with cold, clammy extremities, sunken eyes, dry tongue, thready (irregular) pulse, drawn and anxious face (Hippocratic facies).The patient finally lapses into unconsciousness. With early diagnosis and adequate treatment, this condition is rarely seen in modern surgical practice[18].

SIGNS OF PERITONITIS

Inspection

There is diminution or absence of abdominal respiratory movement. The position of patient in bed is characteristic. He lies still in bed with legs drawn up in an effort to relieve the tension on the abdominal muscles. There is uniform distension of abdomen and in early cases marked retraction of lower half of abdomen. The movements of the abdomen in relation to respiration appear to be restricted.

Palpation

Tenderness and rigidity will be elicited. Tenderness is a constant but not a reliable sign as rigidity. Tenderness is first situated over the causative focus, but spreads with a diffusion of the peritoneal inflammation, which rapidly becomes generalized, and extreme in degree.

There are two other signs that are constantly present:

- Rebound tenderness.
- Pain experienced over the affected region by pressure on an uninvolved region.

Of all signs, rigidity of the abdominal muscles is the most important and reliable sign.

Voluntary guarding following involvement of parietal peritoneum by inflammation, also by reflex spasm may be initially present. As peritonitis advances reflex spasm may become so severe that board like rigidity of abdominal wall is produced.

Percussion

Abdomen is resonant everywhere and resonant tympanic owing to the fact that the intestines are filled with gas. In certain instances, like the perforation of GIT, obliteration of liver dullness is evident. Shifting dullness indicates free fluid within the abdomen.

Auscultation

Bowel sounds are diminished from the onset. They may be absent over the area of greatest mischief, and in all established cases of peritonitis with ileus, there is often a sinister silence[18].

INVESTIGATIONS OF PATIENT WITH PERITONITIS

A number of diagnosis may elucidate doubtful diagnosis, but in the diagnosis, the clinician should rely on history and physical findings mainly.

Routine Investigations

Hemoglobin and urine analysis are done. ESR may be raised, particularly in abdominal tuberculosis affecting the peritoneum. Leukocytosis is usually seen, especially the differential counts with shift to left, are more important.

Peritoneal diagnostic aspiration

It may be useful when sufficient peritoneal fluid is in the peritoneal cavity to be aspirated. First described by Solomon, it is done in four quadrants after infiltrating the skin with a local anesthetic. When aspiration fails, the introduction of a small quantity of sterile physiological saline, followed by aspiration after a few minutes, may produce fluid of diagnostic value. Microscopy of the fluid may show neutrophils more than 250cells/mm3 (indicator of inflammation) and bacteria (indicator of infection). Fluid is also examined for cell count, differential, PH and gram stain and aerobic and anaerobic culture[17].

An erect X-ray film of the abdomen

The X-ray should include the diaphragm, lower chest and pelvis. There may be pneumoperitoneum (demonstrated by gas under the dome of diaphragm) ground glass appearance, obliteration of peritoneal pad of fat line and psoas shadow due to edema of peritoneum. There may be dilated gas-filled loops of bowel (consistent with paralytic ileus). Demonstration of pneumoperitoneum is seen in excess of 70% of cases of GIT origin. If the patient is too ill to stand, lateral decubitus posture can be used.

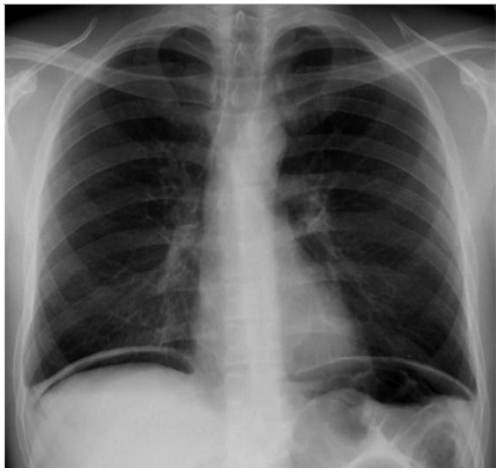
. 

FIG 4 X ray chest showing air under diaphragm

BIOCHEMICAL INVESTIGATIONS

- Estimation of serum electrolytes.
- Serum amylase levels to exclude acute pancreatitis provided it is remembered that moderately raised values are frequently found following other abdominal catastrophes and operations. For e.g., perforated peptic ulcer, Cholecystitis.
- Widal test in ileal perforation to rule out typhoid.

- Blood urea, serum creatinine to know the status of renal system
- Peritoneal fluid for culture and sensitivity: This can be done by aspiration or from fluid derived at laparotomy. It may be particularly helpful in the diagnosis of primary peritonitis.
- Laparotomy is done to diagnose and to treat peritonitis. On laparotomy, the peritoneal cavity can be cleaned by lavage.
- Biopsy can be taken wherever found necessary[18].

Ultrasound and CT scanning

These investigations may also be useful in some patients in identifying the cause of the peritonitis. E.g. perforated appendicitis, acute pancreatitis and also may show fluid collection in peritoneal and pelvic cavities. It may also influence operative approach or contraindicate operation. Other investigations have to be done according to the specific etiology, which is described below.

MANAGEMENT OF PERITONITIS

Standard Treatment

Kirschner, in 1926, formulated two surgical principles for the management of peritonitis which later have become the gold standard[26].

1. " *Plugging* " the source of infection.
2. " *Purging* " the peritoneal cavity of bacteria, toxins and adjuvant.

Thus the laparotomy, repair of bowel leak and peritoneal toilet became the standard therapy, but the morbidity and mortality continued to be high.

Disadvantages of standard operative treatment

This results in tight closure of the abdomen, where intra-abdominal pressure is already high, causing respiratory embarrassment, ventilation perfusion imbalance and its consequences. Sepsis elimination cannot be confirmed with the single laparotomy and there is no control over the intraabdominal process like anastomosis healing or bowel viability.

New operative concepts

The era of new operative concept started in 1975 with the dissertation of Pujol from Parries University. He concludes that intraabdominal Sepsis should be treated like many abscesses in the body. He advocated leaving the abdomen open (laparostomy) and treating like an open wound - A radically different

approach. After this a number of surgeons published their experience with this new operative modality confirming definite improvement in mortality.

GENERAL CARE OF THE PATIENT

Fluid resuscitation

Consists of correction of circulating volume and electrolyte imbalance. Extensive peritoneal inflammation causes fluid to shift into the peritoneal cavity and the intestinal space. Urine output has to be maintained about 30ml/hr. The plasma volume must to be restored and the plasma electrolyte concentration has to be maintained. Central Venous catheterization and pressure monitoring may be helpful in correcting fluid and electrolyte balance particularly in patients with concurrent disease. Plasma protein depletion may also need correction as the inflamed peritoneum leaks large amounts of protein. If the patient's recovery is delayed for more than 7-10 days, parenteral nutrition is required.

Gastrointestinal decompression

A nasogastric tube is passed into the stomach and aspirated. Aspiration is continued until the paralytic ileus has recovered. The amount of fluid draining and the colour sholud be noted- they could be poiniting at the diagnosis in these patients.

Analgesia

Freedom from pain allows early mobilization. Pain in preop setting should be expertly managed. Synthetic opoids can be given- the risk of respiratory depression is low. Adequate physiotherapy in the post-operative period helps to prevent basal pulmonary collapse, deep vein thrombosis and pulmonary embolism.

Vital system support

If septic shock is present, special measures may be needed for cardiac, pulmonary and renal support. Oxygen is administered to overcome the mild hypoxemia that is commonly present in peritonitis because of increased metabolic demands of infection, some degree of intrapulmonary arterio-venous shunting and the mechanical impairment of pulmonary ventilation by a distended, tender abdomen. Ventilatory support should be initiated whenever any of the following are present;

1. Inability to maintain adequate alveolar ventilation as evidenced by a rising PaCO2 of 50 mm Hg or greater.
2. Hypoxemia reflected in PaO2 < 55 mm Hg.
3. Evidence of shallow, rapid respiration due to muscular tiring or the use of accessory muscles of respiration.

Antibiotic therapy

The bacterial flora is monomicrobial in nature, in primary peritonitis and polymicrobial in secondary peritonitis Altemier et al 1938[27]. When experimental peritonitis with E. coli and B. fragilis was treated with different antibiotic regimens, clear patterns of response were seen. Treatment with gentamicin alone improved the acute death rate in the model but had no impact on the abscess phase of the disease. Nicholas et al demonstrated improvement in the death rate of rats with polymicrobial experimental peritonitis induced with a large inoculum, by the addition of clindamycin coverage for B. fragilis. From these animal studies, combination therapy was born and became the standard for the treatment of peritonitis during the late 1970s. In the 1980s, the emergence of single antibiotics with both aerobic and anaerobic activity leads to numerous clinical studies that compared the newer antibiotics to combination therapy. With one exception, most comparative studies consistently demonstrated comparable results with single agent compared to the combination. Costs and drug toxicity reduced with the single antibiotic approach.

As the infection is usually a mixed one, a single or combination therapy that have activity against aerobic and anaerobic bacteria, is used. Culturing peritoneal fluid and modifying the antibiotic subsequent to the culture sensitivity may not always influence the outcome.

Suggested antimicrobial agent therapy for the Treatment of established secondary bacterial Peritonitis

Mild to moderate intra-abdominal infection

Second or third generation cephalosporin OR

ß- Lactamase inhibitor combination OR

Monobactum + metronidazole

Severe intra-abdominal infection without renal dysfunction

Carbapenem OR

Fluoroquinolone + metronidazole OR

Aminoglycosides + metronidazole + ampicillin

Severe intra-abdominal infection with renal dysfunction

Carbapenem OR

Fluoroquinolone + metronidazole[17]

Specific treatment of the cause (operative management)

The primary therapy in the management of generalized peritonitis is surgical. This depends on the cause of generalized peritonitis e.g. perforation closure in case of perforated duodenal ulcer. Though there are other factors that affect the outcome in suppurative peritonitis, timing of operation is an important variable that is often overlooked. In peritonitis due to pancreatitis or salphingitis or in cases of primary peritonitis of streptococcal or pneumococcal

origin, non-operative management is preferred (if the diagnosis is made with certainty).

OPERATIVE PRINCIPLES

1. Control of source of infection- Repair/Plug
2. Purge- Peritoneal lavage and toilet i.e. evacuate bacterial inoculums, pus and adjuvant.
3. Decompress- Treat or avoid intraabdominal compartmental syndrome.
4. Control- Prevent or treat persistent and recurrent infect ion or verify both and purge[26].

Principle –1 repair

The infectious material leaking into the abdomen is to be eliminated. This involves procedures like appendicectomy, closure of duodenal or ileal perforation, resect ion of gangrenous viscera or necrosectomy of pancreas. The bowel ends may be anastomosed, exteriorized or simply closed.

Principle-2 purge

Infectious peritoneal fluid, pus, necrotic tissue and adjuvant either contain bacteria or promote their growths and they should be removed. A large quantity of saline about 8-10 liters may be required for wash and “radical debridement”.

However, too aggressive debridement should be avoided to prevent excessive blood loss or bowel injury. Antibiotic/ betadine wash have not been proved to be any great advantage. At the end no irrigation fluid should be left in the abdomen.

Principle-3 decompresses

During acute peritonitis more than 10 liters of inflammatory fluid may accumulate in the peritoneum and its sub-mesothelial loose connective tissue. The co-existent paralytic ileus, fluid accumulation in the peritoneal cavity, post resuscitation visceral and parietal edema increases the intraabdominal pressure producing a compartment syndrome. In this situation, if the abdomen is closed with tension, there will be impairment of cardiovascular, respiratory, renal and hepatic functions and also splanchnic blood flow and oxygenation. The answer to this problem lies in open abdomen or staged abdominal repair (STAR).

Principle-4 control

This principle aims at having control over the intra-abdominal processes like anastomotic healing, proper closure of perforation, and viability of bowel segments and formation of pus inside the abdomen. This aim is not achieved by the standard operation. This principle allows for frequent re-exploration and peritoneal toilet if required.

NEW OPERATIVE METHODS

With the entire above complex and interesting knowledge, we can now concentrate on the new operative methods evolved for the treatment of severe intra-abdominal sepsis. In 1993, the "International society of surgery" called several experts in this field to the "International surgical week" held at Hong Kong and decided on four basically different methods[26].

- OPA- Open abdomen (Laparostomy)
- COLA- Covered Laparostomy
- PR- Planned relaparotomy
- STAR- Staged abdominal repair

Open abdomen (laparostomy)

This is defined as laparotomy without re-approximation and suture closure of abdominal fasciae and skin. Abdominal cavity is left open like an open wound and dressed and finally heals by granulation. This method takes care of principles- repair, purge and decompression. The disadvantages are, there is no control over intraabdominal process, exposed viscera may perforate and huge ventral hernia results since definitive closure is not possible. Hence it has lost its popularity.

Covered laparostomy (COLA)

This is defined as laparotomy without re-approximation and suture closure of abdominal fasciae and covering the facial gap with materials like merles or vicryl mesh. The viscera may also be covered with skin with relaxing incision.

Planned repaparotomy (PR)

In this approach abdomen is left open initially and re-explored at an interval of 12-24 hours for irrigation, debridement etc. Devices used to ease re-exploration include commercially available Zipper, Ethizip, Velcro, artificial burr, PTFE mesh (Gortex) etc. this procedure allows for having control over intra-abdominal processes.

Staged abdominal repair (STAR)

This is a series of planned abdominal operations with staged re-approximation and final suture closure of the abdominal fasciae. It is planned either before or during the first operation called Index Star. The abdomen is closed temporarily with devices like Zip, Velcro etc. and controlled tension is exerted to the fascia avoiding and intra-abdominal pressure effects. Re-laparotomies are performed at 24 hour intervals at operating room. Once problem is solved abdominal cavity is formally closed.

Indications for STAR

1. Diffuse peritonitis in critical patient condition.

2. Severe peritoneal edema.

3. Source of infection is not controlled.

4. Incomplete debridement of necrotic tissue.

5. When viability of bowel is uncertain, anastomosis / repair needs Re-inspection

6. Uncontrolled bleeding with packing.

7. Infected pancreatic necrosis.

8. Massive abdominal wall loss.

9. Any intra-abdominal problem that is difficult or impossible to manage with a single operation[28].

Advantages of STAR

Staged abdominal repair technique allows for complete repair, debridement and purge. Anastomotic healing is monitored and any complications diagnosed early & corrected. Intra-abdominal compartment syndrome and its consequences are prevented. With the STAR technique colostomies may be avoided in favor of anatomists, abdominal drains with their disadvantages are avoided and finally this technique allows for suture closure of abdomen with sound healing.

Peritoneal lavage

Price first advocated washing the contaminated peritoneal with large volumes of irrigant in 1905. In 1906, Torek reported that large volume irrigation reduced mortality in generalized peritonitis following appendicitis in 14%. Lavage is done on the basis that phagocytic macrophages and neutrophils cannot function unless attached to peritoneal Serosa. They cannot function if they are swimming as phagocytes already dislodged from peritoneum are either dead or non-functional, in which case lavage causes no harm.

There are 3 basic principles of peritoneal lavage

1. To wash the digestive enzymes, that might have leaked into the peritoneal cavity.
2. To remove material like pus, blood and faeces that could harbor or nourish bacteria.
3. To potentiate the antibiotic effect by allowing the topical application of relatively high dosage of these agents.

The majority of surgeons lavage until the fluid is clear, use more than 1 l. In the case of the dirty abdomen (i.e. gross pus or faecal peritonitis), saline, aqueous betadine, water and antibiotic lavage can be used. Surgeons also use IOPL during clean cases.

Drains

The use of drains, particularly sump suction drains is an important aid in the surgical management of intra-abdominal abscesses or similarly localized collection.

CONSERVATIVE MANGEMENT

Conservative management may be advisable in following conditions

- Appendicular abscess when the infection is definitely localized and mass is subsiding.
- Gonococcal peritonitis
- Chronic pelvic abscess
- In primary primary peritonitis of children
- Moribund patients.

COMPLICATIONS OF PERITONITIS

Systemic complication of peritonitis

1. Bacteremic/endotoxic shock
2. Bronchi pneumonia/respiratory failu re
3. Renal failure
4. Bone marrow suppression
5. Multisystem failure

Bacteremic / endotoxic shock

It is due to large amount of exudation from the inflamed peritoneum into the peritoneal cavity, vomiting and paralytic ileus, where the absorbing function of bowel is lost. It depends on the microbial infection in severity. Gram-negative septicemic shock is common in enteric and large bowel perforation.

Bronchopneumonia/ respiratory failure

This occurs in early stage of peritonitis, which is severe. Hurried breathing in early stages is due to under-ventilation, which is because of abdominal distension causing restriction of diaphragmatic and intercostal muscle movement.

Renal failure

Hypovolumia decreased cardiac output, increased secretion of ADH and aldosterone and raised intra-abdominal pressure act together in peritonitis, on the kidney. This is especially true in septic shock. Acute tubular necrosis can occur because of decreased flow and will lead to oliguria and metabolic acidosis.

ABDOMINAL COMPLICATIONS OF PERITONITIS

1. Adhesional small bowel obstruction

2. Paralytic ileus

3. Recurrent or residual abscess

4. Portal pyemia/liver abscess.

Adhesional small bowel obstruction

The adhesions, when fine and minimal, are absorbed, but when dense cause intestinal obstruction at a later date. They manifest with all signs of obstruction. Failure of conservative treatment necessitates surgery, to divide the adhesions and relieve the obstruction.

Paralytic ileus (Neurogenic obstruction)

The bacterial toxins act on neuromuscular junctions and smooth muscle of bowel producing paralytic ileus. It is beneficial as it avoids spreading of the peritoneal contents from perforated viscous to other regions but prolonged paralytic ileus may prove to be a serious setback because fluid loss from the intestine into the lumen may play a large part in protein, water and electrolyte depletion.

Abscess

Presentation may be very vague and consist of nothing more than a lassitude, anorexia, pyrexia (often low-grade), tachycardia, leukocytosis and

localized tenderness. Later on a palpable mass may develop. When palpable, an intra-peritoneal abscess should be monitored by marking out its limitations on the abdominal wall and meticulous examination. Abdominal ultrasound has been a popular method for the diagnosis of intra-abdominal abscess. It is a low cost method. Several radionuclide scans have been developed to identify abscess within the peritoneal cavity. The gallium citrate-67 scan achieved a certain level of popularity for the diagnosis of intra-abdominal abscess. Gallium concentrates within inflammatory foci and with use of radioactive isotope of gallium, a gamma camera should be able to identify collections of pus. More recently, indium 111-tagged leukocytes have been used as another potential imaging technique.

The diagnostic method of choice for abdominal abscesses is CT scan. The CT scan provides remarkable anatomic resolution of normal structures and of abnormal collections of fluids and pus. The use of intraluminal and in some cases, intravascular contrast agents permits differentiation of intraluminal and extraluminal collections. Abscess cavities commonly have air bubbles that augment the judgment that any fluid collection may be an abscess. The accuracy of the CT scan in the diagnosis approaches 90%.In the majority of the patients, with the aid of antibiotic treatment the abscess or mass becomes smaller and smaller and finally is undetectable. In others, the abscess fails to resolve or becomes larger, in the event of which it must be drained. In many situations, the

abscess becomes adherent to the abdominal wall, so that it can be drained without opening the general peritoneal cavity. Other modes of treatment are percutaneous drainage and open drainage of the abscess. Septic patients with evidence of severe clinical infection will usually require open laparotomy and drainage. A persistent septic response with hyperglycemia, gastrointestinal ileus, blood culture positive for anaerobic and enteric pathogens and early evidence of respiratory failure as the initial expression of multi organ failure cascade, mean that a source of clinical infection must be identified and treated.

CLASSIFICATION OF INTRAABDOMINAL INFECTIONS

1. PRIMARY PERITONITIS

a. Spontaneous peritonitis in children.

b. Spontaneous peritonitis in adults.

c. Peritonitis in patients with CAPD.

d. Tuberculosis and other granulomatous peritonitis.

e. Other forms.

2. SECONDARY PERITONITIS

a) Acute perforation peritonitis (Acute supportive peritonitis)

b) Post-operative peritonitis

c) Post-traumatic peritonitis

3. TERTIARY PERITONITIS

a) Peritonitis without evidence for pathogens.

b) Fungal peritonitis.

c) Peritonitis with low grade pathogenic bacteria.

4 . OTHER FORMS OF PERITONITIS

a. Aseptic/sterile peritonitis.

b. Granulomatous peritonitis.

c. Drug-induced peritonitis.

d. Periodic peritonitis.

e. Lead peritonitis.

f. Hyperlipidemic peritonitis.

g. Foreign-body peritonitis.

h. Talc peritonitis.

5. INTRA ABDOMINAL ABSCESS

a. Associated with primary peritonitis.

b. Associated with secondary peritonitis.

Primary peritonitis

Primary peritonitis is an inflammation of the peritoneum from a suspected extra peritoneal source, often via hematogenous spread. Spontaneous bacterial peritonitis isnow more common in adults than in children and shows no differential sex incidence. Adults with cirrhosis or systemic lupus erythematous have replaced children with nephrosis, formerly the group most commonly affected. Spontaneous peritonitis in adults is seen most commonly in patients with ascites and is a monomicrobial infection. Onset is more insidious in ascitic adults. Most patients complain of abdominal pain and distension, vomiting, lethargy and fever more prominent in children. Diarrhea is typical in neonates, but seldom seen in adults. The clinical picture may be non-specific. Paracentecis is the most useful diagnostic test. Fluid is examined for neutrophil cell count; pH and gram stain should be done a specimen sent for culture.The neutrophil cell count has the highest sensitivity and specificity in making the diagnosis. A neutrophil count > 250 cells / cu mm is positive. Ascitic fluid pH is low in spontaneous bacterial peritonitis. Only one third of patients with positive fluid cultures. If the stain shows only gram-positive cocci, spontaneous peritonitis is strongly suggested; if a mixed flora of gram positive and negative is present, intestinal perforation is more likely. When the diagnosis of spontaneous bacterial peritonitis is confirmed, antibiotic therapy should be started and the patient initially managed nonoperatively.

Secondary peritonitis

Chemical (aseptic) peritonitis

Aseptic peritonitis refers to the peritoneal inflammation from substances other than bacteria. A perforated peptic ulcer provides the most severe and common form of chemical peritonitis with gastric juice and bile contaminating the peritoneal cavity.

Biliary peritonitis alone may follow gangrene and perforation of the gallbladder. Blood in the peritoneum is also a cause of peritoneal irritation after slow bleeding (e.g. a ruptured graafian follicle or following splenic injury) rather than from a catastrophic hemorrhagic event as a ruptured aneurysm where the primary pathology itself overshadows the peritoneal irritation. Meconium and urine may also precipitate chemical peritonitis.

Peritonitis due to perforated peptic ulcer

The perforation generally occurs as sudden, relatively catastrophic event. The patient with a perforated peptic ulcer classically presents with abrupt onset of epigastric pain, with or without radiation to shoulder. Generalized peritonitis supervenes within hours and the patient lies motionless to minimize pain. These classic features may be absent in several circumstances. In very young or aged, immuno suppressed, quadriplegic and comatose patients, perforation may be present in a much more subtle manner. The classic presentation can be modified

when gastric juice flows down the paracolic gutters, simulating acute appendicitis on the right side and acute sigmoid diverticulitis on the left. In the other forms, a perforated duodenal ulcer simulates perforated gall bladder and duodenum[29]. Sometimes, following an ulcer perforation, the ulcer may seal rapidly before there is a spillage of gastric and duodenal contents.

Other rare presentations of perforated duodenal ulcer

1. Perforation associated with hemorrhage is rare but a grave complication. The bleeding arises from erosion of large vessel such as gastroduodenal artery. The clinical picture is that of acute perforation of peptic ulcer with signs of hemorrhage.

2. Perforation and pyloric stenosis, this combination is very rare. Lam and colleagues in 1978 noted that 4 out of 244 patients had this combination of perforation, hemorrhage and obstruction.

3. Retroperitoneal perforation; it usually follows blunt trauma to the abdomen in the epigastric region. It is more difficult to detect. Patient may have pain in the epigastric region and back and may develop vomiting. Later, patient may develop retroperitoneal cellulitis and succumb to it. In still some other cases, the pus may track retroperitoneally into the right iliac fossa and may present as a mass simulating appendicular abscess which on drainage may lead to duodenal fistula.

Upper gastro intestinal study with gastrograffin series

The use of water soluble radio contrast material is advocated in diagnostic work up of the patient with duodenal ulcer perforation. Without pneumoperitoneum it confirms diagnosis, the site, presence of ulcer crater, whether perforation is sealed off or not.

It has some disadvantages like

1. Pylorospasm induced by the water soluble contrast may impair clear visualization of the duodenum.
2. The time taken to perform a contrast study at odd hours.

In retroperitoneal perforation following features may be seen in the erect abdominal X-ray.

- Mild scoliosis, usually concave to the right.
- Obliteration of psoas shadow.
- Retroperitoneal air around upper pole of the right kidney along the right psoas muscle and around the transverse mesocolon.

Treatment

The following treatment has been described for perforated ulcer.

Simple closure of perforation with omental patch.

Definitive treatment for the ulcer at the time of perforation closure

This includes simple closure of perforation with drainage procedures like gastroenterostomy with or without vagotomy.

For gastric perforation four quadrant biopsy has to be taken and if the patient is fit, gastric resection with ulcer has to be done unless the ulcer is juxta esophageal, in which case the ulcer should be repaired and a tanner procedure should be held in reserve as a secondary choice.

Laparoscopic closure of perforation

Appendiceal perforation

Immediate appendicectomy, has long term been the recommended treatment of acute appendicitis because of the known progression to rupture. Studies have shown that delays in presentation were responsible in majority of perforated appendices. There is no accurate way of determining when and if an appendix will rupture prior to resolution of the inflammatory process.

Appendiceal rupture occurs most frequently distal to the point of luminal obstruction along the antimesentric border of the appendix. Rupture should be suspected in the presence of fever greater than 39 C and a WBC count greater than 18000/mm3 . Generalized peritonitis will be present if the walling off process is ineffective in containing the rupture. Thus one has to be very vigilant

of clinical signs and history in detecting the progression of appendicitis- mass vs abscess.

Treatment

Treatment consists of appendicectomy and peritoneal lavage and antibiotics. The skin and subcutaneous tissue should be left open and allowed to heal by secondary intention in 4 to 5 days as delayed primary closure.

Typhoid perforation

Typhoid perforation is usually seen in the third week of infect ion with Salmonella typhi in patients with acute disease. The disease is endemic in regions with poor hygienic conditions. Typhoid bacilli are thought to pierce the Peyer's patches of the intestinal wall, mainly in the distal ileum. These collections of lymphoid cells hypertrophy leading to hemorrhage and then perforation. Perforation often is not appreciated in an already severely diseased patient and it is super infection resulting from leakage of intestinal bacteria that leads to the full-blown picture of suppurative bacterial peritonitis. Widal test will be positive in such patients[15].

Treatment

Surgical Management

At laparotomy, a single perforation is found on the anti-mesentric border of the ileum in 80 per cent of the patients. Two perforations are found in 15 per cent and more than two in 5 per cent. About 90 per cent of ileal perforations are located within 60cm of the ileo-caecal valve and caecal perforations occur in only 2 percent of the patients.

Perforations at the sites other than ileum and caecum are extremely rare. A simple debridement of the margin of the perforation and meticulous closure in two layers with copious peritoneal lavage, is the procedure of choice. However, when there are more than three perforations, which are close together, it is best to resect the affected bowel and perform a primary end-to-end anastomosis. Any areas of apparent impending perforations, if not included in a resection, must be over sewn. A right hemicolectomy is undertaken only for caecal perforations.

Following peritoneal lavage, the abdominal wound is closed, usually without drains. If there is gross faecal contamination, the skin wound may be left open to minimize wound infection. The anti-typhoid drug therapy should be continued for at least 14 days[30].

Operative procedures in typhoid perforation

Procedure Indication	Comments
1. Simple closure	Simple but high leak rate in some series

2. Debridement/ wedge excision + simple closure for single ileal perforation	Simple and effective operation
3. Resections	Extensive operations
Ileal resection + primary anastomosis	For multiple ileal perforations.
4.Right hemicolectomy for caecal perforation	Morbid procedure
5.Simple closure or ileal resection + end-to-side ileotransverse colostomy.	Has decreased morbidity but not mortality.
6. Ileostomy of perforated ileum	In extremely critical or moribund patients.
7. Simple peritoneal drain	In extremely critical or moribund patients
8. Oversewing	For areas of impending perforation

Colonic perforation

Perforation is less common than is obstruction, occurring in about 5 percent of patients. The site of perforation is usually within the tumor and is not associated with obstruction but is the consequence of tumor necrosis. Rapid cardiovascular collapse and endotoxaemic shock, usually signify a major leak and faecal peritonitis. About 22 percent of the cases of peritonitis have their

origin in colon. More than half of these are due to inflammatory diseases, such as diverticulitis. The remaining cases are due to perforation proximal to or at stenosis caused by luminal bowel obstruction (tumor) or external bowel obstruction such as incarcerated hernia, intussusception and volvulus... A malignant growth usually does not cause peritonitis directly but may
lead to bowel obstruction with either perforation of dilated segments or bowel ischemia and/or bacterial migration through the necrotic bowel wall.

Surgical treatment

The goal of operation is to remove the diseased perforated segment of the bowel. It is possible to fashion a primary resection and end-to-end anastomosis. However, an anastomosis of unprepared bowel fashioned in a contaminated field should always be protected by proximal colostomy or ileostomy. The temporary diverting stoma can be closed about ten weeks after the emergency operation.

An alternative is to resect the perforated segment and to exteriorize the proximal and distal loops of the bowel, where the proximal opening acts as the colostomy and the distal as the mucous fistula or to use Hartman's operation for more distal lesions, where the distal end is not possible to be brought to the surface of the abdomen. In the Hartman's operation, the diseased segment is

excised, end colostomy (proximal) and closure of distal stump is done. Anastomosis is done at a later date.

If peritonitis is severe and the patient is not fit for surgery, three stage procedure is preferred. The first stage of the classic three –stage procedure consists of proximal colostomy (transverse). In the second stage, resection of the diseased segment and anastomosis is done. In the third stage, colostomy closure is done. There are considerable drawbacks to the three stage procedure. These include a focus of infection in the abdomen for an unduly longer period before the second stage procedure is done, also the length of time for which transverse colostomy may be present and for the patients to cope with the malodorous fluid effluent from the proximal stoma.

Tuberculous peritonitis

Two forms of peritonitis are seen- Acute and chronic

Acute tuberculous peritonitis

This type has an onset that resembles so closely acute peritonitis that the abdomen is opened straw-colored fluid escapes and tubercles are seen scattered over the peritoneum and greater omentum. Early tubercles are greyish and translucent. They soon undergo caseation, and appear white or yellow and are then less difficult to distinguish from carcinoma. Occasionally, they appear like patchy fat necrosis.

Chronic tuberculous peritonitis

The condition presents with abdominal pain (90%) cases, fever (60%), loss of weight (60%), ascitis (60%), night sweats (37%) and occasionally as abdominal mass.

Origin of infection

Infection originates from;

- Tuberculous mesenteric lymph nodes;
- Tuberculosis of ileocaecal region;
- A tuberculous pyosalphinx;
- Blood borne infection from pulmonary tuberculosis, usually the 'miliary', but occasionally the 'cavitating' forms.

Varieties of tuberculous peritonitis

There are four varieties of tuberculous peritonitis

a. Ascitic.

b. Encysted.

c. Fibrous.

d. Purulent.

Ascitic form

The peritoneum is studded with tubercles and peritoneal cavity becomes filled with pale straw colored fluid. The onset is insidious. Pain is often

completely absent; in other cases there is considerable abdominal discomfort, which may be associated with constipation of diarrhea. On inspection, dilated veins can be seen coursing beneath the skin of abdominal wall. Shifting dullness can be readily elicited.

Encysted form (loculated)

Encysted form is similar to the above, but one part of the abdominal cavity alone is involved. Thus a localized intra-abdominal swelling is produced, which gives rise to difficulty in diagnosis.

Fibrous form (Plastic)

Fibrous form is characterized by the production of wide spread adhesions, which cause coils of intestine, especially the ileum to become matted together and distended. These distended coils act as a 'blind loop' and give rise to steatorrhoea, wasting and attacks of abdominal pain. On examination, the adherent intestine with omentum attached, together with the thickened mesentery, gives rise to a palpable mass. The first intimation of the disease may be sub-acute or acute intestinal obstruction.

The division of bands can remedy sometimes the cause of the obstruction easily. If the adhesions are accompanied by fibrous strictures of the ileum as well, it is best to excise the affected bowel, provided not too much of the small

intestine needs to be sacrificed. If adhesions are only present, a plication may be performed. Chemotherapy after adequate surgery will rapidly cure the condition.

Purulent form

The purulent form is rare, and usually occurs secondary to tuberculous salphingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Sizable cold abscesses often form and are present on the surface, commonly near the umbilicus, or burst into the bowel. In addition to prolonged general treatment, operative treatment may be necessary for the evacuation of the cold abscesses and possibly for the intestinal obstruction. The prognosis of this form of peritonitis is

relatively poor.

Diagnosis

A peritoneal fluid tap will show mostly lymphocytes. Tubercle bacilli can be retrieved from ascitic fluid in 80 percent of the time if more than one liter of fluid is cultured. The ascitic fluid has an increased protein concentration, lymphocytic pleocytosis andglucose concentration below 30mg/dl. At laparotomy a peritoneal biopsy should be taken. The placement of drains or exteriorization of bowel should be avoided.

Treatment

Medical line of management

Anti-tubercular chemotherapy should be instituted in all cases of abdominal tuberculosis. At present, the anti-tuberculosis regimen recommended by W.H.O and The International Union against Tuberculosis and Lung diseases is Isoniazid (300mg daily), Rifampicin (450mg daily), Pyrazinamide (1.5gm daily orally) and Ethambutol (25mg/kg/day) or Streptomycin (0.75gm intramuscularly daily) for two months, followed by Isoniazid(600mg) and Rifampicin (600mg) twice weekly orally for four months for an individual of 40-60 kg body weight. The patient is monitored periodically especially for hepatotoxicity. Pyridoxine hydrochloride (5-10 mg/day) must be given along with Isoniazid to prevent peripheral neuropathy.

Surgical line of management

Operation should be reserved for diagnosis if needle biopsy fails or for treatment of such complications as fecal fistula or obstruction and performed as described earlier.

Management of tuberculous perforations

According to the site of perforation;

• Gastro-duodenal type; closure with ATT.

• Small bowel type; closure with ileo-transverse anastomosis placed proximal to perforation with ATT.

• Large bowel type; Ileo-transverse anastomosis for lesions on right side and proximal colostomy for left -sided lesions with ATT.

Definitive surgery after patient improves.

Amoebic perforation

Entamoeba histolytica infection of the intestine usually causes dysentery like illness, but sometimes liver abscesses or perforation of large bowel occurs. Liver abscesses also can rupture and can cause diffuse peritonitis. The clinical picture is that of bacterial peritonitis. Treatment consists of resection of the diseased bowel segment with anastomosis and, administration of metronidazole in combination with a third generation cephalosporin is carried out[15].

Meconium peritonitis

Meconium is a sterile mixture of epithelial cells, mucin, salts, fats and bile. It is formed when the fetus commences to swallow amniotic fluid. Meconium peritonitis is an aseptic peritonitis, which develops, late in intrauterine life or during or just after delivery. In the remainder no cause for the perforation is discernable. It causes matting of intestinal loops and in some cases, the extruded meconium becomes calcified in a matter of weeks. Meconium remains sterile until about three hours after birth; thereafter, unless

the perforation has sealed, sterile meconium peritonitis gives way to acute bacterial peritonitis, which, unless treated promptly, is rapidly fatal[18].

Foreign body peritonitis

Foreign bodies may be deposited in the peritoneal cavity during operations (sponge or instrument inadvertently left behind) or may result from penetrating injuries or perforation of the intestine following ingestion. A larger foreign body can lead to the formation of an abscess in the presence of bacteria, but otherwise foreign bodies are sealed off and encapsulated.

Periodic peritonitis

Recurrent episodes of abdominal pain, fever, and leukocytosis occur in certain population groups, notably in Americans, Arabs and Jews. The disease appears to be familial. The major point for the surgeons is that, laparotomy is not required in these episodes. Laparotomy is often performed for the first episode, since an acute intra-abdominal process requiring surgical cure cannot be ruled out. At operation, the surfaces may be inflamed and there is free fluid but no bacteria. Colchicine is effective in preventing recurrent attacks and a favorable response to chronic administration of colchicine is a definitive diagnostic test.

Drug related peritonitis

Administration of INH and Erythromycin estolate has been reported to cause acute abdominal symptoms mimicking peritonitis but not development of true peritonitis. A number of cases have been reported in which, beta-blocking drugs haveresulted striking thickening of visceral peritoneum. The most frequent clinical presentation is a typical small bowel obstruction, often subtle at onset associated with weight loss and with an abdominal mass on physical examination. The agglomeration of the small bowel produces the mass that is palpable preoperatively.

Lead peritonitis

Lead peritonitis has the same clinical picture as intermittent porphyria is associated with lead intoxication (occurring in painters, smelter workers, pica in children), and a careful history will lead to correct diagnosis.

Hyperlipedimic peritonitis

Abdominal pain mimicking peritonitis may be seen in patients with type 1 and type V hyper lipoproteinemia a group of heterogeneous disorders resulting from increased concentration of chylomicrons or VLDL in the blood. If erroneously operated on during early stages, the abdominal cavity is found to

be full of chylous milky material. A careful family history will clarify the differential diagnosis.

Porphyric peritonitis

It is seen in patients with acute intermit tent porphyries, who suffer from attacks that cause nervous system damage especially autonomic system. The pain may be localized or generalized and is often accompanied by vomiting and constipation. The diagnosis is established by the demonstration of porphobilinogen in the urine by Watson-Schwartz test.

Talcum peritonitis

Peritoneal inflammation, exudation and formation of pseudo tumor (chronic inflammatory omental tumors) and formation of dense adhesion may follow contamination of peritoneal cavity by glove lubricants (talc, lycodium, mineral oil, corn starch, rice starch) or by cellulose fibers from disposable gauze pads and gowns. The reaction, particularly to rice starch, is largely a hypersensitivity response. When the diagnosis remains unclear, laparoscopy is useful. If the peritonitis is recognized, reoperation may be avoided and corticosteroids or non-steroidal anti-inflammatory drugs administered. Eventually the peritonitis resolves.

Tertiary peritonitis

Patients, in whom peritonitis and sepsis initially have been controlled operatively and in whom bacteria have been eliminated by successful antibiotic therapy, may progress to tertiary peritonitis. It is a state in which, host defense system produces a syndrome of continued systemic inflammation. The clinical picture is one mimicking occult sepsis, as manifested by a hyper dynamic cardiovascular rate, low grade fever and general hyper metabolism. The patient had a clinical picture of sepsis, without the focus of infection. Such patients sometimes are subjected to laparotomy in an attempt to provide drainage of anticipated recurrent or residual collections of infected fluid. On operation, no pathogens are present. Empiric anti-infective therapy is of no value.

Malignant peritonitis (carcinoma peritonii)

This can produce acute and sub-acute peritonitis. It is extremely rare. Primarily, it is a mesothelioma of fibro-sarcomatous nature, which occurs in asbestos workers. Secondary tumor is common mainly from stomach, ovary and large intestine and very rarely from distant sources like breast, lung etc.

Pseudomyxoma peritonei

More frequently in females the abdomen is filled with yellow jelly, large quantities of which are often more or less encysted. The condition is associated

with both mucinous cystic tumors of ovary and appendix. Recent studies suggest that most cases arise from primary appendiceal tumors with secondary implantation on to one or both ovaries. It is often painless and there is frequently no impairment of general health for a long time. If the abdomen seems to be distended with fluid, which cannot be made to shift, it should raise the suspicion of pseudomyxoma peritonei. At laparotomy, masses of jelly may be seen which are scooped out. The appendix, if present, should be excised with any ovarian tumor. Unfortunately, recurrence is common. Pseudomyxoma peritonei is locally malignant, but does not give rise to extra-peritoneal metastasis. Occasionally, the condition responds to radioactive isotopes or intra peritoneal chemotherapy, which may be used in recurrent cases[31].

Post-puerperal peritonitis

Post-puerperal peritonitis, following puerperal infection, is more common after first deliveries. Rigidity is seldom present. This is partly due to stretched condition of the abdominal musculature. The lochia may be offensive but not necessarily so. Diarrhea is common.

Treatment

If the infection is strictly limited to the pelvis, the correct treatment is to rest the gastrointestinal tract and provide intravenous fluid, antibiotics and

correct the electrolyte imbalance. Posterior colpotomy for pelvis abscess can be done.

Peritonitis related to peritoneal dialysis

• Peritonitis is the dominant complication of continuous ambulatory peritoneal dialysis (CAPD), in patients in end-stage renal disease.

• Peritonitis occurs more frequently with CAPD than with intermittent Peritoneal dialysis.

• Catheter related infection is the most common mechanism. Other causes of peritonitis in CAPD are tunnel infections and cuff extrusion.

• Two-thirds of the patients with positive cultures have a gram-positive coccus as the positive organism, usually Staphylococcus aureus or Staphylococcus epidermidis. Turbidity of the dialysate is the earliest and the only finding in one-fourth of the cases.

The diagnosis is established when any of the following are present;

a. Positive culture from the peritoneal fluid.

b. Cloudy dialysate effluent.

c. Clinical signs of peritonitis.

Treatment

The initial treatment is administration of antibiotics and heparin in the dialysate as well as an increase in the dwell time of dialysate fluid. The indication for catheter removal include, persistence of peritonitis after 4 to 5 days of treatment, the presence of fungal or tubercular peritonitis, faecal peritonitis or severe skin infection at the catheter site[15]. Post-operative period was monitored; intake output charts and vital charts were maintained.

Drains were removed after 48 hours and sutures were removed on the 7th post-operative day. Most of the operated patients had uneventful recovery. Diagnosis is confirmed by histopathology reports. The patients were followed up for a variable period of time.

NEED FOR SCORING SYSTEMS

In this present era where there is an increasing demand to audit the quality of surgical care provided by surgeons, scoring systems may provide a measure of differentiating surgeon dependent and independent variables.

The preoperative risk calculated by these scoring systems may also help in prognosticating patients[19] and decide on the course of further management operative vs non operative, damage control vs definitve surgery etc. Arriving at a preoperative risk may also help in communicating with the patient's side better about the condition and expected outcome[32].

New techniques of intervention may be tried in different risk groups. These may then be compared in RCT to decide upon which is best suited for a particular risk group. Diffuse peritonitis continues to have a high mortality rate inspite of intensive care. Therefore the need to correctly identify this subset for appropriate management.

EXISTING SCORING SYSTEMS

	Scores predicting mortality	Scores predicting morbidity
Scores not requiring operative information	ASA APACHE-II Sickness Assessment Boey Score Hacetteppe Score Physiological POSSUM	APACHE-II Veltkamp Score VA Pneumonia Prediction Index VA Respiratory Failure Score
Scores requiring operative Information	Mannheim Peritonitis Index Reiss Index Fitness Score POSSUM, P-POSSUM Cleveland Colorectal Model Surgical Risk Scale	POSSUM, P-POSSUM

MANNHEIM PERITONITIS INDEX

Mannheim peritonitis index was developed by Wacha and Linder in 1983 based on retrospective evaluation of 1253 patients with peritonitis. 20 variables were identified of which 8 were found to influence the outcome and was considered to be prognostically relevant. They were incorporated into the score. MPI is a relatively simple score and easier to arrive at than other lab dependent investigations and the information needed for calculating the score is usually found in surgical registers[9].

A detailed study of MPI was done by A. Billing[20] in 7 different centers and their data compared. They considered patients of perforated or postoperative peritonitis, peritonitis caused by pancreatitis, appendicitis and mesenteric ischemia for study. Each risk factor is given a weightage to produce a score used for prognostic purposes. They found linear correlation between mean index score and mean mortality rate.

- Maximum score is 47
- The cutoff point taken was a score of 26. Patients with higher values being classified as non-survivors.
- Patients were divided into 3 categories of severity.

MPI < 21, 21 – 29, > 29.

Advantage of MPI

- It is one of the easiest scores to apply
- The determination of risk is available during operation
- Surgeon can know about the possible outcome and the appropriate management can be decided. Patient with less score can be treated with usual minimal risks, while patient with high score may need aggressive approach with critical care monitoring. Concept of programmed relaparotomy, zip technique surgery may need to be considered in these cases.
- It is peritonitis specific index and appears to be the best for statistical studies and comparing clinical trials. Other scores like Apache-II score are not specific for peritonitis.

Disadvantages

- This index does not include the possibility of eradicating the source of inflammation.
- It is a one time score; hence post-operative complications may hamper the results.
- The index assigns peritonitis originating from colon to be a low risk. Since most of the colonic performances are usually secondary to malignancy, this may not be applicable uniformly.

MANNHEIM PERITONITIS INDEX

Name: **Age:** **Sex:** **IP NO:** **S.NO**

DIAGNOSIS:

PROCEDURE:

1. Age>50 years **(5)** _____

2. Female sex **(5)** _____

3. Organ failure **(7)** _____

Creatinine level	>177 umol/L
Urea level	>167 mmol/L
Oliguria	<20 ml/h
Lung PO2	<50 mmHg
PCO2	>50 mmHg
Paralytic ileus	>24h
Mass in	
USG /CT/Per abdominal examination	
Per rectal examination	

4. Malignancy **(4)** _____

5. Preoperative duration of peritonitis>24 hrs **(4)** _____

6. Origin of sepsis not colonic **(4)** _____

7. Diffuse generalized peritonitis **(6)** _____

8. Exudate

Clear **(0)** _____

Cloudy/ purulent **(6)** _____

Faecal **(12)** _____

TOTAL _____

p-POSSUM

The Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM) was developed by multivariant discriminant analysis of 48 physiological and 18 operative variables by Copeland et al in 1991[33]. Eighteen of these variables were determined to be independent factors related to patient outcome. POSSUM has since been shown to overpredict mortality in low risk patient groups. The Portsmouth predictor equation (p-POSSUM) was developed to overcome this failing[34]. Variations of this score include Cr-POSSUM for colorectal malignancies and V-POSSUM for vascular case[35].

POSSUM scoring

Physiological and operative severity score for the enumeration of mortality based on Copeland, Jones and Walters Br J Surg(1991).Scores are calculated taking into consideration 2 parameters

Physiological severity

Age, cardiac signs, respiratory signs, systolic blood pressure, pulse, Glasgow coma scale, hemoglobin, total count, urea, sodium, potassium and ECG.

Operative severity

Multiple procedures, total blood loss, peritoneal soiling, malignancy, operative severity and mode of surgery.

It is considered to be midway between too simple ASA scoring and too complex APACHE II scoring system.

Drawbacks

Tends to overestimate the mortality in low risk patients[36].

Tends to overestimate if used in other specialties.

P POSSUM –Portsmouth predictor equation for mortality

Prytherch et al Br J Surg 1998[37] introduced the corrected version of the scoring system. This scoring is more accurate than the original POSSUM scoring but it still overestimates the mortality in low risk patients. Higher the risk more is the accuracy of the scoring system. There have been new versions of this scoring system like V-POSSUM used specifically for specialties.

Predicted death rate=$1/ (1+ e^{-R})$

Where R is (0.1692×physiological score) + (0.1550×oerative score) -9.065 in POSSSUM

R = (0.13×physiogical score) + (0.16 ×operative score)-7.04 in P-POSSUM.

p-POSSUM SCORE

Name: **Age:** **Sex:** **IP NO:** **S.NO**

DIAGNOSIS:

PROCEDURE:

PHYSIOLOGICAL SCORE

PARAMETER	OBSERVED VALUE	SCORE
AGE		
CARDIAC SIGNS		
RESPIRATORY HISTORY		
SBP		
PULSE RATE		
GCS		
HEMOGLOBIN		
WBC COUNT		
SERUM UREA		
SERUM SODIUM		
SERUM POTASSIUM		
ELECTROCARDIOGRAM		
TOTAL SCORE		

OPERATIVE SCORE

PARAMETER	OBSERVED VALUE	SCORE
OPERATION SEVERITY		
REOPERATION		
PERITONEAL SOILING		
MALIGNANCY		
BLOOD LOSS		
URGENCY OF SURGERY		
TOTAL SCORE		

$$\text{Ln}[R/(1-R)] = -9{\cdot}065+(0{\cdot}1692\times \text{PSS})+(0{\cdot}155\times \text{OSS})$$

p-POSSUM SCORE= _____

APACHE-II

APACHE-II is based on APACHE -I which was develoed by Knaus et al[22] in 1983.Originally developed for an ICU application, this score has been embraced by the surgeons owing to its accuracy in prediction. The APACHE II (Acute Physiology and Chronic Health Evaluation) score integrates estimates of the severity of disease measured by 12 physiological variables with the physiological reserve estimated by age and chronic disease score[23]. The original APACHE was designed to predict mortality risk for stratification by assessing the patient independent of effects of treatment (surgery). It was primarily designed to stratify ICU admissions[24].

Disadvantages

- The primary intra op findings are not considered in the data collected. These data have a significant bearing on the outcome of the patient. These are especially important if the APACHE was calculated after the primary surgery. The primary surgery would alter the physiological variables used to calculate the APACHE score.
- Mortality prediction is less accurate
- It is difficult to use this scoring system for postoperative monitoring because of the need to measure numerous laboratory parameters when deciding therapies for patients with unsuccessful initial treatment

- The acquisition of so many physiological variables will always make it less readily measured than other scoring systems.
- This initial stratification of risk factors and a predicative equation estimate patient outcome. They are, however, both complex and time consuming and provide a one time estimate. However, Continuous APACHE scoring has been developed to overcome this[38].

APACHE- II

Name: **Age:** **Sex:** **IP NO:** **S.NO**

DIAGNOSIS:

PROCEDURE:

A.AGE SCORE:

B.ACUTE PHYSIOLOGICAL SCORE

PHYSIOLOGICAL VARIABLE	OBSERVED VALUE	SCORE
Temperature^{O}C		
MAP (mm of hg)		
Heart rate		
Respiratory rate		
PaO2 (mm of Hg)		
Arterial Ph		
Serum Na (mmol/L)		
Serum K (mmol/L)		
Serum creatinine (mg/dl)		
Hematocrit		
WBC(cells/cu mm)		
Serum HCO_3		
Serum urea(mg/dl)		
GCS		
TOTAL SCORE		

C. CHP (Non operative or emergency postoperative -5 points / Elective postoperative -2 points.)

_____

Documented cirrhosis/ Portal hypertension/ UGI bleed Hepatic failure/ encephalopathy/ coma NYHA class IV symptoms/ inability to climb stairs/ do household chores Documented hypoxia/ hypercapnia/ secondary polycythemia On immunosuppressive therapy/ on high dose steroids/ CT/ RT/ leukemia/ lymphoma/ HIV

APACHE SCORE(A+B+C)=_____

SEPSIS SCORE OF ELEBUTE AND STONES

It was first published in 1983. It was primarily designed for district general hospitals, for monitoring patients suffering from peritonitis. The authors divided the clinical features of the septic state into 4 classes to which they ascribed a subjective degree of severity on an analogue scale. The attributes were local effects of tissue infection, degree of temperature elevation, secondary effects of sepsis and laboratory data[25]. This system was examined in more detail by Dominioni in 135 patients. The sepsis scores range from 10 to > 30. The overall accuracy was 84% for mortality.

Advantages

1. Since, this was primarily designed for district hospitals, it is more appropriate for Indian set up.
2. Since, it includes detailed clinical work up, it is more sensitive.
3. The range of lab tests is kept minimum.
4. It can be used either as a single one time score or can be used to monitor critical patients and score tabulated on regular basis.

Disadvantages

1. Most of the attributes are calculated subjectively, hence more prone for observer variations.

2. No direct attempt to score “septic shock”, hence it provides indirect evidence for sepsis syndrome.

Other scoring systems

ASA SCORING

This is in spite of its subjective nature and the inter-observer variation in measuring has been used for many years, and remains the score routinely used in most surgical emergency cases. It was not designed to predict mortality but it has been shown to give a good estimate of mortality risk with the great advantage of being simple to score. It is subjective and may be applied inconsistently by different anesthetists. The fact that ASA scores vary between observers suggests that it is really an expert clinical assessment of risk and not a score at all.

BOEY SCORING SYSTEM

- Shock at admission (systolic blood pressure <90 mmHg),
- medical illness (ASA III–V), and
- Delayed presentation (duration of symptoms >24 h).

ADVANTAGES

Simple , easy to remember and apply.

DISADVANTAGES

Does not consider various other physiological factors which do have a significant role in predicting the patient condition. and it is less accurate.

HACETEPPE SCORE USED IN PEPTIC ULCER PERFORATION

There are four variables in the study

- the presence of a serious coexisting medical illness,
- acute renal failure,
- white cell count of more than 20×10^9/l, and
- male sex.

VELTKAMP SCORE

Eleven patient, disease and surgery-related variables are used. Minor complications are less-successfully predicted hence it is less commonly used.

VA RESPIRATORY FAILURE PREDICTION INDEX

The VA study was modelled on over 80, 000 men who developed respiratory failure (defined as mechanical ventilation for 48 hours or more) after (non-cardiac) surgery.

WHICH SCORING IS BEST?

Though no major studies have been conducted to compare all the studies, as most of the system requires different clinical and laboratory parameters, almost all researchers agree for a reliable, simple and easily reproducible scoring system which helps not only in decision making, prognosticating and grading sepsis but also can be used for comparing data at different institutes.

Billing[20] who conducted study of MPI in 2003 patients at different centers in three different European countries reported that is not only reliable in predicting mortality but can also be used for comparative study.

Demmel[12] conducted a study comparing MPI with Apache-II scores, and concluded that both scores were equally accurate, but MP1 was easier and disease specific.

Pacelli[1] conducted study in 1996, comparing MPI, Apache and sepsis score. They concluded that MP1 and Apache-II correctly predicted death as outcome, but MP1 was easier to calculate.

Ohmann.C[21] concluded none of the existing score was of particular use for therapeutic decision making in peritonitis. The new prognostic model should be the focus of further trials.

Deducing from above studies, it appears that MPI and sepsis score seem to be appropriate study for patients with peritonitis and sepsis syndrome, in a district hospital set up as it utilizes minimum investigations and can be used for

predicting outcome of the patient. The score conceived by John Boey with reference to perforated duodenal ulcers can be utilized for decision making as regard to what surgery is to be performed whether to operate at all.

MATERIALS AND METHODS

1. **DESIGN OF STUDY** : A Prospective Analytical Study done in Govt Rajaji Hospital, Madura1i, Tamil Nadu,
2. **PERIOD OF STUDY** : One Year from September 2013 to November 2014
3. **COLLABORATING DEPARTMENT** :Department of Biochemistry, Madurai Medical College.
4. **SELECTION OF STUDY SUBJECTS** : All patients admitted in General surgical wards of Govt. Rajaji Hospital with perforative peritonitis were included in the study after getting informed written consent.
5. **DATA COLLECTION** :Required data were collected from the complaints, history of presenting illness and past history of the patient , radiological investigations, biochemical lab values, intraoperative findings.
6. **ETHICAL CLEARANCE** : Approved by the Institute of Ethical Comimttee, Madurai Medical College.
7. **CONSENT** :Informed written consent from the patient obtained in the patient's mother tongue.
8. **ANALYSIS**: All data were analysed using SPSS Version 15 for Windows software. Area under the curve was calculated using Receciver operator characteristic curves.
9. **CONFLICT OF INTEREST** : NIL
10. **FINANCIAL SUPPORT :** NIL

11. **PARTICIPANTS:** Patients diagnosed with Perforative pertonitis were enrolled after getting informed written consent.

12. **INCLUSION CRITERIA:** All patients admitted in General surgical wards of Govt Rajaji Hospital with perforative peritonitis were included in the study which includes hollow viscous perforation due to peptic ulcer disease, enteric fever, trauma- blunt or penetrating, cases of intestinal obstruction with strangulation and ruptured liver abscess.

13. **EXCLUSION CRITERIA:** Pateints with spontaneous peritonitis, age< 12 years and those with postoperative peritonitis were excluded from the study.

14. **METHODOLOGY:** After the relevant data were collected in printed proforma sheets containing the requisite variables necessary, they were entered into online score calculators (www.SFAR.org and www.riskpredicton.org.uk). The calculated scores were tabulated and analysed using statistical software SPSS.

15. **DEFINITIONS**

MORTALITY all deaths within 30 days of surgery were taken into account.

DISCRIMINATORY ABILITY otherwise defined as accuracy- ability of a test to discriminate with precision those who are at risk of dying and those who are not.

POSITIVE PREDICTIVE VALUE is defined as the proprtion of the patients with postive test who have the disease.

SHARPNESS OF PREDICTION is the ability of the test to assign subjects to wither of the outcome groups.

RELIABILITY of the scores is assessed by comparing the observed mortality with expected mortality obtained from other studies.

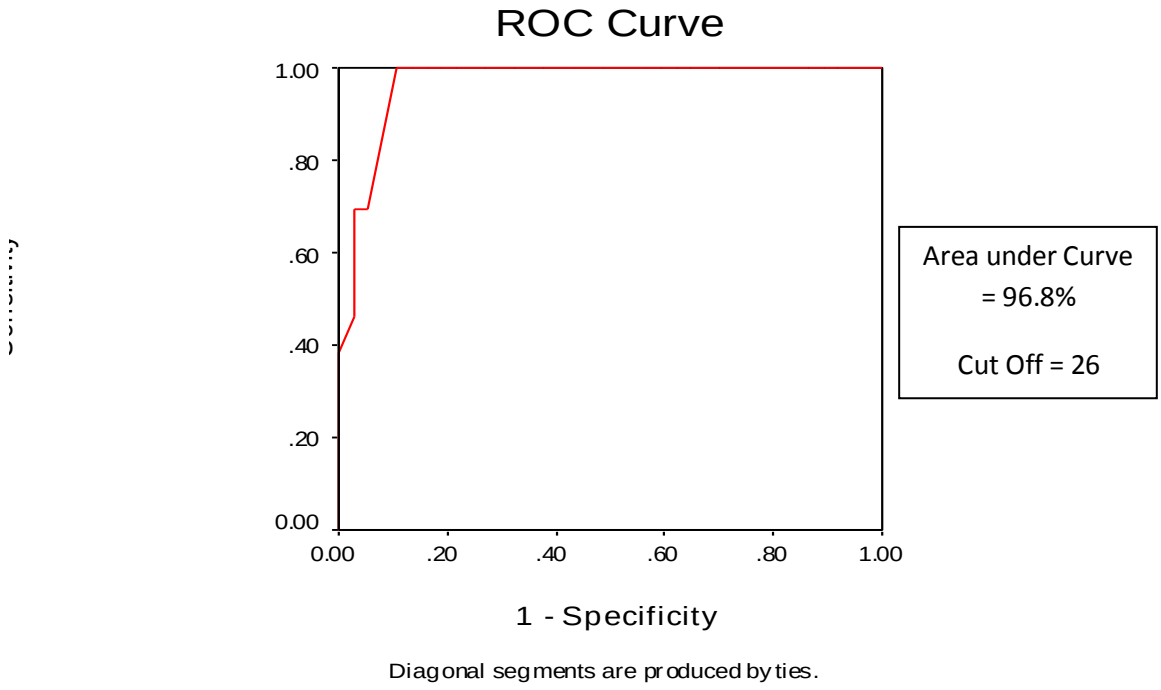
OBSERVATIONS AND RESULTS

DISCRIMINATORY ABILITY AND CUT OFF POINTS

Discriminatory ability or accuracy was analyzed using ROC and area under the curve was calculated

Cut Off Point for MPI

ROC analysis was done to identify the best cut off for MPI. The cut off that we got was 26 for which the sensitivity and specificity was calculated to be 100% and 89.19% respectively.



GRAPH 1- ROC curve for MPI

From the ROC curve, the area under the curve for MPI was calculated as 96.8% which is statistically significant .

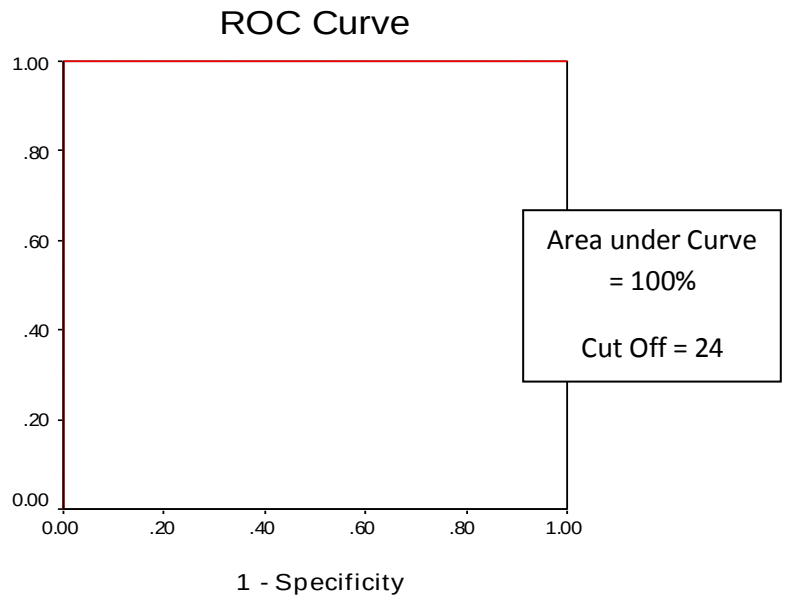
TABLE 1- ANALYSIS OF MPI SCORING SYSTEM WITH CUT-OFF OF 26

Indices	MPI
Sensitivity	100 %
Specificity	89.19%
Positive Predictive Value	76.47%
Negative Predictive Value	100%
Positive Likelihood Ratio	9.25
Negative Likelihood Ratio	0

The positive predictive value of MPI was 76.47%

Cut Off Point for APACHE-II

ROC analysis was done to identify the best cut off for APACHE-II. The cut off obtained was 24 at which the sensitivity and specificity was calculated to be 100% and 100% respectively.



GRAPH 2 -ROC curve for APACHE-II

From the ROC curve, the area under the curve for APACHE-II was calculated as 100% which is statistically significant .

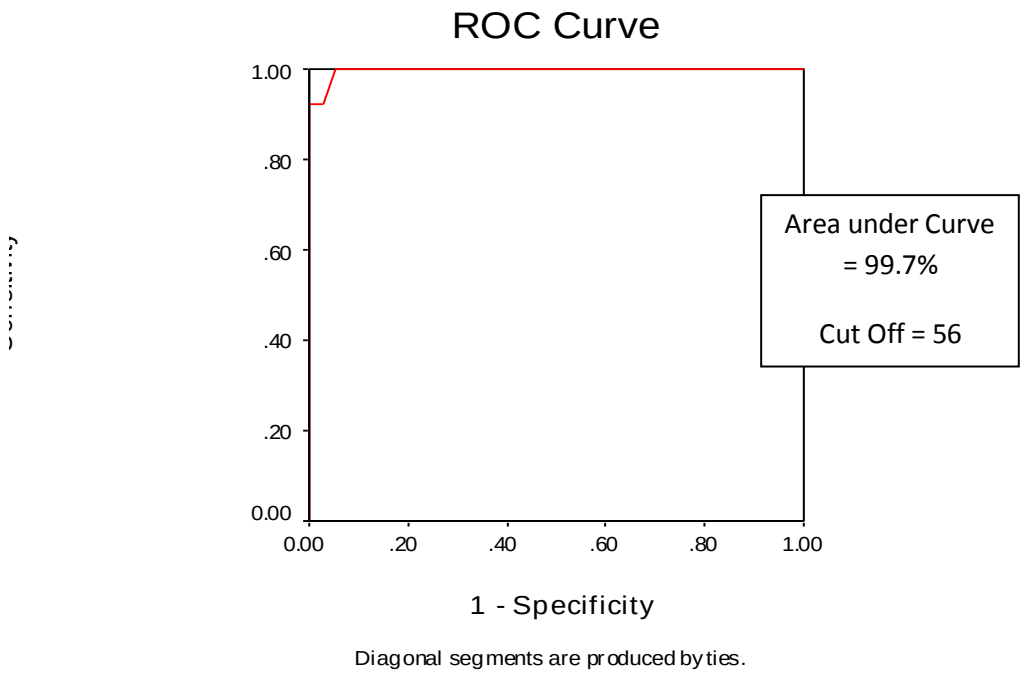
TABLE 2- ANALYSIS OF APACHE-II SCORING SYSTEM WITH CUT-OFF OF 24

Indices	APACHE-II
Sensitivity	100 %
Specificity	100%
Positive Predictive Value	100%
Negative Predictive Value	100%
Positive Likelihood Ratio	-
Negative Likelihood Ratio	0

The positive predictive value was 100% for APACHE-II.

Cut Off Point for p-POSSUM

ROC analysis was done to identify the best cut off for p-POSSUM. The cut off was found out to be 56 at which the sensitivity and specificity was calculated to be 100% and 94.59% respectively.



GRAPH 3- ROC curve for p-POSSUM

From the ROC curve, the area under the curve for p-POSUM was calculated as 99.7% which is statistically significant .

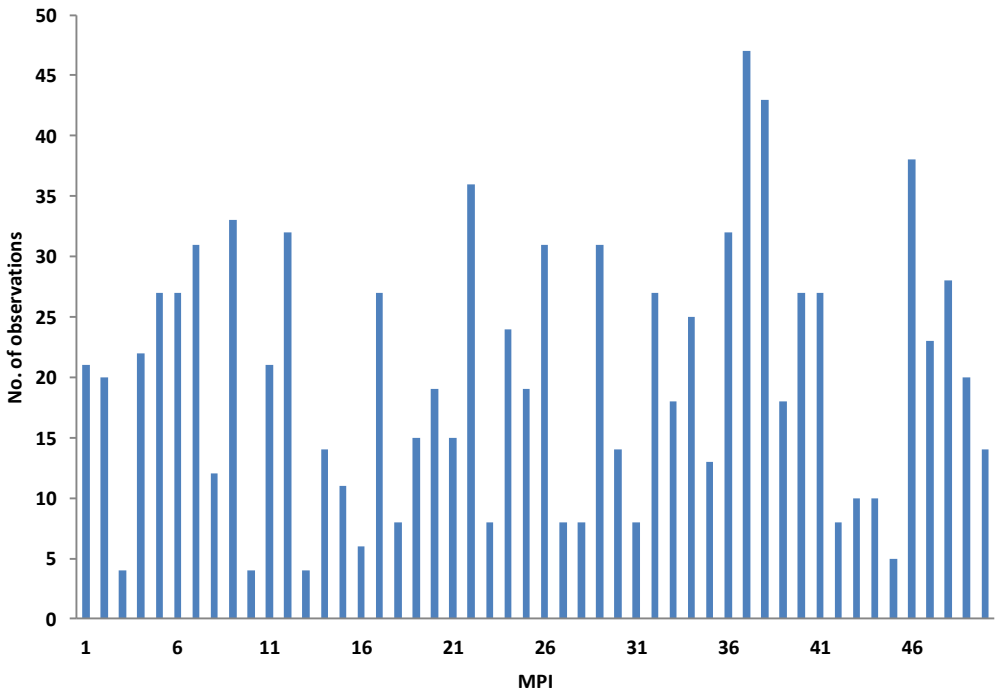
TABLE 3- ANALYSIS OF P-POSSUM SCORING SYSTEM WITH CUT-OFF OF 56

Indices	p-POSSUM
Sensitivity	100 %
Specificity	94.59%
Positive Predictive Value	86.67%
Negative Predictive Value	100%
Positive Likelihood Ratio	18.50
Negative Likelihood Ratio	0

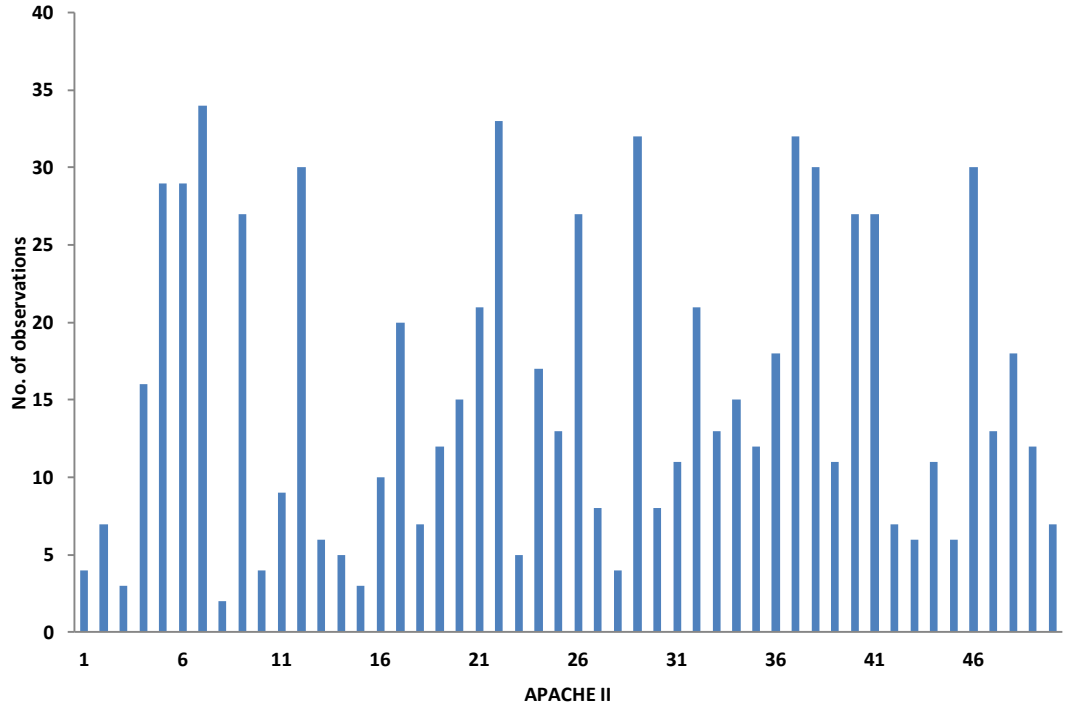
The positive predictive value was 86.67% for p-POSSUM.

SHARPNESS OF PREDICTION

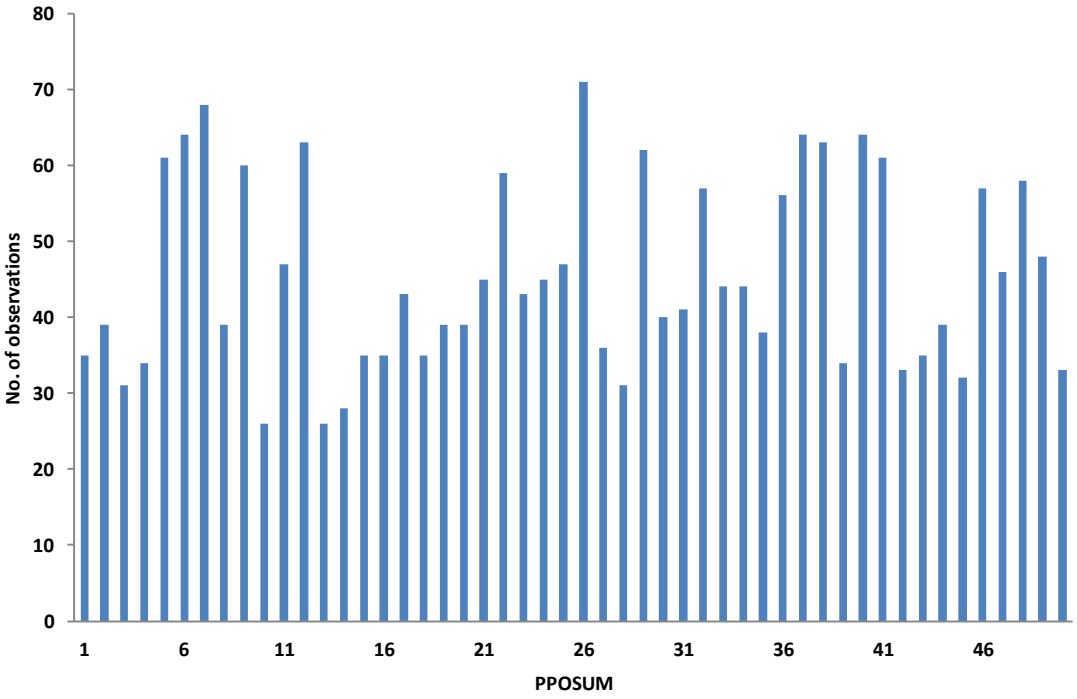
The following graphs plot the scores obtained against the number of observations with the score in the x-axis and y-axis respectively for Mannheim peritonitis index, APACHE-II and p-POSSUM scores respectively. Sharpness is defined as the ability of a test to predict with accuracy any one outcome- in this case either a low probability of death < 0.1 or an increased probability of death .0.9.



GRAPH 4- FREQUENCY OF OBSERVATIONS IN MPI



GRAPH 5- FREQUENCY OF OBSERVATIONS IN APACHE-II



GRAPH 6- FREQUENCY OF OBSERVATIONS IN p-POSSUM

TABLE 4- SHARPNESS OF PREDICTION

Score	Probability of death		
	<0.1 (Sharp)	0.1-0.89 (Not sharp)	≥0.9 (Sharp)
APACHE II	17	33	-
p-POSSUM	24	25	1

Probabilities are calculated from the predicted mortality percentages. Number of observations for probability < 0.1 and probability > 0.9 are calculated. We find that both of them show similar sharpness of prediction. In APACHE-II 51.51% of values were within the above parameters and in p-POSSUM 50%of values were within the set parameters.

RELIABILITY OF PREDICTION

On comparing the mortality rate with similar studies we get the following results

TABLE 5 Mortality rate by MPI scoring system

Score	Number of patients	Deaths	Mortality Rate
≤ 20	27	0	0%
21 – 29	13	4	30.8%
≥ 30	10	9	90%

TABLE 6 Comparison of MPI with other studies

MPI score	**Ermolov *et al.*, (1996)**	**Quereshi *et al.*, (2005)**	**Present study**
< 21	0%	1.9%	0%
21-29	42%	21.9%	30.8%
> 30	100%	28.1%	90%

One can safely assume there is some degree of variation among prediction of mortality in different score classes in different studies.

TABLE 7 Mortality rate by APACHE-II scoring system

Score	Number of patients	Deaths	Mortality Rate
≤ 10	19	0	0%
11 – 20	16	0	0%
>20	15	13	86.7%

TABLE 8 Comparison of APACHE-II with other studies

APACHE-II	**Ahmad& Malik et al**	**Ahuja & Pal**	**Present Study**
< 10	0%	0%	0%
11-20	35.3%	18.75%	0%
>20	95.7%	50%	86.7%

APACHE-II also shows results similar to MPI.

TABLE 9 Mortality rate by p-POSSUM scoring system

Score	Number of patients	Deaths	Mortality Rate
≤ 35	15	0	0%
36 – 55	19	0	0%
>55	16	13	81.3%

DISCUSSION

DISCRIMINATORY ABILITY AND DETERMINATION OF CUT OFF POINTS

Receiver operator characteristic curves[39] were used to calculate the discriminatory ability of each of the scores. It is a graph plotted between sensitivity and 1- specificity. The area under the curve (AUC) for each of the scores was calculated for different cutoff points and the cut off at which maximum AUC was obtained was chosen.

TABLE 10- AUC & CUT OFF

S.no	Name of the scoring system	AUC	Cut off
1	MPI	96.8%	26
2	APACHE-II	100%	24
3	p-POSSUM	99.7%	56

APACHE-II had the maximum area under the curve followed by p-POSSUM and MPI. APACHE-II is a perfect test that has the capability to predict with maximum accuracy the subset of patients that are going to die from perforative peritonitis. p-POSSUM comes a close second with an area of 99.7% and MPI is third with a score of 96.8%.

Various studies have identified the optimal cut off point for MPI as 26[40,41,42]. This study gets a similar result.ROC studies reveal that the cut off for APACHE should be fixed at 24 for maximum results. p-POSSUM was found to have a cut off of 56.

POSITIVE PREDICTIVE VALUE

The positive predictive values for the three scores are as follows.

TABLE 11- POSITIVE PREDICTIVE VALUE

S.no	Name of the scoring system	Positive Predictive Value
1	MPI	76.47%
2	APACHE-II	100%
3	p-POSSUM	86.67%

APACHE-II easily trumps the other two with a positive predictive value of 100%.

SHARPNESS OF PREDICTION

This is defined as the proportion of predictions that come under an outcome, either mortality or low probability of death[43]. MPI is not analysable on a continuous scale[43]. This fact has been documented by some studies. So on analysing the remaining two slides, we can conclude that both APACHE and p-POSSUM are similar in sharpness (50% vs 51.51%)

RELIABILITY OF PREDICTION

On comparing the observed mortality values for each of the scores with mortality obtained from other studies, we find that there is disagreement among risk predictions in different class groups in both MPI and APACHE-II.

Analysis of individual scores

MPI

This score has the lowest positive predictive value and discriminatory ability of the three. Regardless, as scores increase over the set cutoff, MPI is accurate[44] in predicting mortality as depicted in Table no . Numerous studies have placed MPI on par with APACHE-II in predicting mortality[41] though this study fails to find such an association, the difference in AUC between the two being statistically significant.

The advantages of MPI have been highlighted before. The inconsistency of MPI may probably be attributed to the fact that it does not take into account all physiological derangements and also that colonic perforations are given less weightage. The probabilities of death obtained in the study correlate well with expected mortalities obtained from other studies. Its safe to assume that MPI is fairly reliable. As scores go beyond the set cut off, mortality increases[45].

APACHE-II

APACHE-II has the maximum AUC. The cutoff point obtained in the study seems to be a little on the higher side compared to previous studies. APACHE-II is accurately able to predict death despite not taking into account intra operative findings and the underlying pathology[38]. There is a definite discrepancy between studies elsewhere and this study in probabilities of death

for patients with score 11-20. One also has to remember that APACHE-II scores have never been used for individual patients and they always have been applied for groups.

p-POSSUM

The main advantage of POSSUM unfortunately is also its Achilles heel- its dependence on intra operative findings. While one may assume that its accuracy of prediction may be enhanced by this characteristic, it also makes it less useful in a preoperative setting. p-POSSUM performed admirably running APACHE-II a close second in discriminatory ability[36]. The cut off obtained in this study is very high compared to cut off values from similar studies[46]. Like APACHE it assigns a lot of intermediate probabilities and therefore is not a very sharp score. As one approaches the higher scores the probability of death shows an increase that corresponds well with expected mortality. p-POSSUM is less difficult to calculate than APACHE-II and as if not more reliable.

CONCLUSION

MPI is an easily calculable, score with good discriminatory ability and reliability let down by a moderate positive predictive value. The cut off obtained was 26[44] which is comparable to other studies. This score is apt for hospitals in a peripheral setting as it does not overemphasize on intensive monitoring and biochemical values[20].

APACHE-II is a highly accurate score with good reliability at higher scores and moderate sharpness. The cut off obtained is 24 which is reasonably similar to cut off values obtained elsewhere. The main difficulty in computing this score is the plethora of biochemical and hematological values needed. Of the three APACHE had the highest positive predictive value.

p_POSSUM is easily relatable to a surgeon and is nearly as accurate as APACHE-II. The cut off value obtained in this study did not match similar studies. The sharpness of this study is comparable to APACHE-II. p-POSSUM is known to over predict mortality particularly in those with a high risk; a significant proportion of the sample presented late and with significant co morbidities. This accounted for a good number of cases with a more risk and high scores and probably is responsible for the high cut off value.

To conclude we can say that while these scores do provide a method of estimating mortality, they are no substitute to clinical management. None of the scores here provide a dynamic assessment of the patient as they are a calculated

only once at a particular point of time. APACHE-II seems to be the ideal score; still using more than one score may improve the sharpness and reliability of prediction.

ANNEXURES

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MANNHEIM PERITONITIS INDEX

Name: **Age:** **Sex:** **IP NO:** **S.NO**

DIAGNOSIS:

PROCEDURE:

1. Age>50 years **(5)** _____

2. Female sex **(5)** _____

3. Organ failure **(7)** _____

Creatinine level >177 umol/L
Urea level >167 mmol/L
Oliguria <20 ml/h
Lung PO2 <50 mmHg
PCO2 >50 mmHg
Paralytic ileus >24h
Mass in
USG /CT/Per abdominal examination
Per rectal examination

4. Malignancy **(4)** _____

5. Preoperative duration of peritonitis>24 hrs **(4)** _____

6. Origin of sepsis not colonic **(4)** _____

7. Diffuse generalized peritonitis **(6)** _____

8. Exudate

Clear **(0)** _____

Cloudy/ purulent **(6)** _____

Faecal **(12)** _____

TOTAL _____

p-POSSUM SCORE

Name: **Age:** **Sex:** **IP NO:** **S.NO**

DIAGNOSIS:

PROCEDURE:

PHYSIOLOGICAL SCORE

PARAMETER	OBSERVED VALUE	SCORE
AGE		
CARDIAC SIGNS		
RESPIRATORY HISTORY		
SBP		
PULSE RATE		
GCS		
HEMOGLOBIN		
WBC COUNT		
SERUM UREA		
SERUM SODIUM		
SERUM POTASSIUM		
ELECTROCARDIOGRAM		
TOTAL SCORE		

OPERATIVE SCORE

PARAMETER	OBSERVED VALUE	SCORE
OPERATION SEVERITY		
REOPERATION		
PERITONEAL SOILING		
MALIGNANCY		
BLOOD LOSS		
URGENCY OF SURGERY		
TOTAL SCORE		

$$\text{Ln}[R/(1-R)] = -9{\cdot}065+(0{\cdot}1692\times \text{PSS})+(0{\cdot}155\times \text{OSS})$$

p-POSSUM SCORE= _____

Key

Physiological Severity Score (PSS)	Operative Severity Score(OSS)
Age (4)	Operation severity (4)
Cardiac signs (4)	Number of procedures (3)
Respiratory history (4)	Blood loss (4)
Systolic blood pressure (4)	Peritoneal soiling (4)
Pulse rate (4)	Malignancy (4)
Glasgow Coma Score (4)	Urgency of surgery (3)
Haemoglobin (4)	
White cell count (3)	
Serum urea (4)	
Serum sodium (4)	
Serum potassium (4)	
Electrocardiogram (3)	

Values in parentheses are the number of severity grades allocated to each score. POSSUM, Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity; p-POSSUM, Portsmouth predictor equation.

APACHE- II

Name: **Age:** **Sex:** **IP NO:** **S.NO**

DIAGNOSIS:

PROCEDURE:

A.AGE SCORE:

B.ACUTE PHYSIOLOGICAL SCORE

PHYSIOLOGICAL VARIABLE	OBSERVED VALUE	SCORE
Temperature^{O}C		
MAP (mm of hg)		
Heart rate		
Respiratory rate		
PaO2 (mm of Hg)		
Arterial Ph		
Serum Na (mmol/L)		
Serum K (mmol/L)		
Serum creatinine (mg/dl)		
Hematocrit		
WBC(cells/cu mm)		
Serum HCO_3		
Serum urea(mg/dl)		
GCS		
TOTAL SCORE		

C. CHP (Non operative or emergency postoperative -5 points / Elective postoperative -2 points.)

_____

Documented cirrhosis/ Portal hypertension/ UGI bleed Hepatic failure/ encephalopathy/ coma NYHA class IV symptoms/ inability to climb stairs/ do household chores Documented hypoxia/ hypercapnia/ secondarypolycythemia On immunosuppressive therapy/ on high dose steroids/ CT/ RT/ leukemia/ lymphoma/ HIV

APACHE SCORE(A+B+C)=_____

Key

	Abnormal Range (High)						Abnormal Range (Low)		
Physiologic variable	**+4**	**+3**	**+2**	**+1**	**0**	**+1**	**+2**	**+3**	**+4**
Temperature - rectal (˚c)	≥41	39-40.9		38. 5-38 .9	36-38. 4	34-35.9	32-33 .9	30-31. 9	≤29.9
Mean Arterial Pressure-mm Hg	≥160	130-159	110-129		70-109		50-69		≤49
Heart Rate (ventricular response)	≥180	140-179	110-139		70-109		55-69	40-54	≤39
Respiratory Rate- (non ventilated or ventilat ed)									
Oxygenation: A. a DO_2 or PaO_2 (mm Hg) a. FIO_2 ≥0.5 record A. a DO_2	≥500	350-499	200-349		≤200				
b. FIO_2 < 0.5					PO_2 <70	$PO_2$6 1-70		PO_2 55-60	PO_2<55
Arterial p^H	≥7.7	7.6-7.69		7.5-7.59	7.33-7.49		7.25-7.32	7.15-7.24	≤7.15
Serum Sodium (mMol/L)	≥180	160-179	155-159	150-154	130-149		120-129	111-119	≤110
Serum Potassium (mmol/L)	≥7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		≤2.5
Serum Creatinine (Mg/100 ML)	≥3.5	2-3.4	1.5-1.9		0.6-1.4		0.6		

White Blood Count (total/mm^3) (in. 1000)	≥40	20-39.9	15-19.9	3.1-4.9	1.2.9		≤
Glasgow Coma Score (GCS) : Score = 15 minus actual GCS							
Total Acute Physiology Score (APS) Sum of the 12 Individuals Variable Points							
SERUM HCO_2 (venous mMol/L) (Not preferred, use if no ABGs)	≥52	41-51.9	32-40.9	22-31.9	18- 21.9	15-17. 9	≤15

A. Age[Yrs] Points	**B. Chronic health points**	**C. Cardiovascular**:	**APACHE II SCORE:**
≤44 0	With history of severe organ insufficiency or immunosuppression assign points as follows:	New York Heart Association Class IV. **Respiratory**: Chronic restrictive, obstructive or vascular disease	Sum of (A)+(B)
45-54	2a. Nonoperative or emergency	resulting in severe exercise	(+C)
55-64	postoperative -5 points	restriction, i.e., unable to climb	
	3b. Elective postoperative -2	upstairs or perform household	A: APS
65-74	points.	duties; or documented chronic	points
5	**Definitions**	hypoxia, hypercapnia, secondary	
≥75	Organ insufficiency or	polycythemia, severe pulmonary	B: Age
6	immunocompromised state	hypertension [.40mmhg], or	points
	must have been evident prior to	respiratory dependency.	
	this hospital admission and	**Immunocompromised**: the	C:
	conform to following criteria:	patient has received	Chronic Health
	Liver Biopsy proven cirrhosis	immunosuppressive therapy	points
	and documented portal	[chemotherapy, radiation long	
	hypertension, episodes of upper	term or recent high dose steroids]	
	GI bleeding or prior episodes of	or has disease that is sufficiently	
	hepatic	advanced to suppress immunity	
	failure/encephalopathy/coma	e.g. AIDS, Lymphoma,	
		leukemia.	

MASTER CHART

S.no	IP No	Age/Sex	MPI	APACHE-II	APACHE-II% Mortality	P-POSSUM Physiologic score	p-POSSUM operative score	p-POSSUM % Mortality	Outcome	Diagnosis
1	34507	50/M	21	4	5.10	16	19	3.19	Recovered	Pre-pyloric perforation
2	6225	28/M	20	7	7.60	20	19	6.08	Recovered	Jejunal perforation
3	38979	15/M	4	3	4.40	16	15	1.74	Recovered	Appendicular perforation
4	40630	75/M	22	16	23.50	21	13	2.94	Recovered	Duodenal perforation
5	42041	35/M	27	29	67.20	38	23	71.69	Expired	Ileal perforation
6	7420	45/M	27	29	67.20	41	23	80.80	Expired	Jejunal perforation
7	36650	38/M	31	34	81.00	41	27	88.66	Expired	Perforative Peritonitis with septicemia
8	8170	22/M	12	2	3.80	13	26	5.54	Recovered	Stab- Colonic perforation
9	46294	45/M	33	27	60.50	30	30	65.95	Expired	Colonic perforation
10	39992	14/M	4	4	5.10	12	14	0.77	Recovered	Intestinal obstruction ileal stricture
11	43976	47/M	21	9	9.90	16	31	17.47	Recovered	Duodenal perforation
12	45458	59/M	32	30	70.30	36	27	77.05	Expired	Sigmoid volvulus
13	40141	34/M	4	6	6.70	15	11	0.80	Recovered	Appendicular perforation
14	41567	42/M	14	5	5.80	15	13	1.09	Recovered	Duodenal perforation
15	5448	29/M	11	3	4.40	22	13	3.46	Recovered	Stab-jejun perforation
16	5447	48/M	6	10	11.30	18	17	3.28	Recovered	Stab-tran col perforation
17	31713	45/M	27	20	35.50	21	22	10.89	Recovered	Ileal gangrene
18	4269	19/M	8	7	7.60	14	21	3.10	Recovered	Stab-jejunal perforation
19	2810	57/M	15	12	14.60	18	21	5.93	Recovered	Stab-ileal perforation
20	23149	55/M	19	15	21.00	17	22	5.85	Recovered	Duodenal perforation
21	1790	65/M	15	21	38.90	23	22	14.64	Recovered	Blunt trauma-Transverse colon perforation
22	32669	60/M	36	33	78.60	31	28	62.71	Expired	Gastric perforation
23	31057	40/M	8	5	5.80	21	22	10.89	Recovered	Duodenal perforation
24	20630	16/M	24	17	26.20	23	22	14.63	Recovered	Duodenal perforation
25	33709	60/M	19	13	16.50	25	22	19.38	Recovered	Jejunal perforation

26	23609	45/M	31	27	60.50	45	26	96.29	Expired	Gastric perforation
27	23638	28/M	8	8	8.70	20	16	3.91	Recovered	Duodenal perforation
28	6076	19/M	8	4	5.10	15	16	1.71	Recovered	Duodenal perforation
29	619	50/M	31	32	76.00	35	27	73.92	Expired	Gastric perforation
30	87848	35/M	14	8	8.70	21	19	7.13	Recovered	Intestinal obstruction ileal stricture
31	85955	40/M	8	11	12.90	22	19	8.33	Recovered	Ileal perforation
32	75071	34/M	27	21	38.90	34	23	56.29	Recovered	Small Bowel gangrene
33	44017	55/M	18	13	16.50	22	22	12.64	Recovered	Duodenal perforation
34	36830	37/F	25	15	21.00	25	19	13.12	Recovered	Intestinal obstruction post operative band
35	21163	17/F	13	12	14.60	20	18	5.26	Recovered	Ileal stricture -post TB
36	19780	46/F	32	18	29.10	29	27	50.67	Recovered	Intestinal obstruction Band
37	45582	65/F	47	32	76.00	36	28	79.67	Expired	Small Bowel malignancy
38	18391	60/F	43	30	70.30	35	28	76.79	Expired	Large Bowel obstruction
39	37990	54/F	18	11	12.90	18	16	2.82	Recovered	Gall Bladder perforation
40	24213	24/M	27	27	60.50	41	23	80.80	Expired	Duodenal perforation
41	21553	40/M	27	27	60.50	38	23	71.69	Expired	Duodenal perforation
42	18843	50/M	8	7	7.60	15	18	2.32	Recovered	Duodenal perforation
43	17343	49/M	10	6	6.70	17	18	3.23	Recovered	Ileal perforation-Typhoid
44	32336	45/M	10	11	12.90	20	19	6.08	Recovered	Ruptured liver abscess
45	38761	35/F	5	6	6.70	18	14	2.08	Recovered	Ruptured ovarian cyst
46	20098	73/M	38	30	70.30	33	24	77.79	Expired	Ca colon Obstruction with peritonitis
47	10870	32/M	23	13	16.50	29	17	20.52	Recovered	Stab- Liver Laceration
48	55255	54/M	28	18	35.50	31	27	59.03	Recovered	Duodenal perforation
49	49233	40/M	20	12	12.90	26	22	22.17	Recovered	Pre pyloric perforation
50	30967	38/M	14	7	7.60	15	18	2.32	Recovered	Appendicular perforation

MANNHEIM PERITONITIS INDEX

S. no	Pt name	IP no	Age>50	Female	Sr.Cr>177 µmol/L	Urea>167 mmol/L	Oliguria < 20 ml/hr	pO2<50 mm of Hg	pCO2>50 mm of Hg	P.Ileus >24 hrs	Mass abd in USG /CT/PR/PA	Malig nancy	Duration > 24 hrs	Origin not colonic	Diffuse	Exudate	MPI Score	Outcome
1	Subbaiyan	34507	No	No	No	No	No	No	No	Yes	No	No	Yes	NC	No	Cloudy	21	Recovered
2	Rajendran	6225	No	No	No	No	No	No	No	No	No	No	Yes	NC	Yes	Cloudy	20	Recovered
3	Tamilselvam	38979	No	No	No	No	No	No	No	No	No	No	No	NC	No	Clear	4	Recovered
4	Veeranan	40630	Yes	No	No	No	No	No	No	Yes	No	No	No	NC	Yes	Clear	22	Recovered
5	Chinna murugan	42041	No	No	Yes	Yes	Yes	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	27	Expired
6	Krishnnan	7420	No	No	No	Yes	No	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	27	Expired
7	Ravi	36650	No	No	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	NC	Yes	Cloudy	31	Expired
8	Arun Arockiaswamy	8170	No	No	No	No	No	No	No	No	No	No	No	C	No	Faeculent	12	Recovered
9	Ganesan	46294	No	No	No	No	No	No	No	Yes	Yes	Yes	Yes	C	Yes	Faeculent	33	Expired
10	Ganesh	39992	No	No	No	No	No	No	No	No	No	No	No	NC	No	Clear	4	Recovered
11	Deivendran	43976	No	No	No	No	No	No	No	Yes	No	No	Yes	NC	No	Cloudy	21	Recovered
12	Lingupandi	45458	Yes	No	Yes	Yes	Yes	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	32	Expired
13	Karthick	40141	No	No	No	No	No	No	No	No	No	No	No	NC	No	Clear	4	Recovered
14	Marimuthu	41567	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Cloudy	14	Recovered
15	Kannan	5448	No	No	No	No	Yes	No	No	No	No	No	No	NC	No	Clear	11	Recovered
16	Rajendran	5447	No	No	No	No	No	No	No	No	No	No	No	C	No	Cloudy	6	Recovered
17	John Peter	31713	No	No	No	No	No	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	27	Recovered
18	Karthick Kumar	4269	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	8	Recovered
19	Mariappan	2810	Yes	No	No	No	No	No	No	No	No	No	No	NC	No	Cloudy	15	Recovered
20	Meeramoideen	23149	Yes	No	No	No	No	No	No	No	No	No	Yes	NC	No	Cloudy	19	Recovered
21	Valasuthurai	1790	Yes	No	No	No	No	No	No	No	No	No	Yes	C	No	Cloudy	15	Recovered
22	Arockiam	32669	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	NC	Yes	Cloudy	36	Expired
23	Veluswamy	31057	Yes	No	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	8	Recovered
24	Anandamurugan	20630	No	No	No	No	No	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	24	Recovered
25	Periaswamy	33709	Yes	No	No	No	No	No	No	No	No	No	Yes	NC	Yes	Clear	19	Recovered

26	Arumugam	23609	No	No	No	Yes	No	No	No	Yes	No	Yes	Yes	NC	Yes	Cloudy	31	Expired
27	Kuzhanthairaj	23638	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	8	Recovered
28	Ilayaraja	6076	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	8	Recovered
29	Ponnasi	619	No	No	Yes	Yes	Yes	No	No	Yes	No	Yes	Yes	NC	Yes	Cloudy	31	Expired
30	Ramachandran	87848	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Cloudy	14	Recovered
31	Raja	85955	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	8	Recovered
32	Appadurai	75071	No	No	No	No	No	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	27	Recovered
33	Pappathy	44017	Yes	Yes	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	18	Recovered
34	Parameswari	36830	No	Yes	No	No	No	No	No	No	No	No	Yes	NC	Yes	Cloudy	25	Recovered
35	Indirakumari	21163	No	Yes	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	13	Recovered
36	Jothi	19780	No	Yes	No	No	No	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	32	Recovered
37	Chellammal	455832	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	NC	Yes	Faeculent	47	Expired
38	Panchali	18391	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	C	Yes	Faeculent	43	Expired
39	Pappathy	37990	Yes	Yes	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	18	Recovered
40	Muthiah	24213	No	No	Yes	Yes	Yes	Yes	Yes	Yes	No	No	Yes	NC	Yes	Cloudy	27	Expired
41	Subbiah	21553	No	No	Yes	Yes	Yes	No	No	Yes	No	No	Yes	NC	Yes	Cloudy	27	Expired
42	Palaniswamy	18843	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Clear	8	Recovered
43	Pandian	17343	No	No	No	No	No	No	No	No	No	No	No	NC	No	Cloudy	10	Recovered
44	Karuppuswamy	32336	No	No	No	No	No	No	No	No	No	No	No	NC	No	Cloudy	10	Recovered
45	Mallika	38761	No	Yes	No	No	No	No	No	No	No	No	No	NC	No	Clear	5	Recovered
46	Nagarajan	20098	Yes	No	No	No	Yes	No	No	Yes	Yes	Yes	Yes	C	Yes	Faeculent	38	Expired
47	Jegan	10870	No	No	No	No	Yes	No	No	No	No	No	No	NC	Yes	Cloudy	23	Recovered
48	Alagupandi	55255	Yes	No	No	No	No	No	No	Yes	No	No	No	NC	Yes	Cloudy	28	Recovered
49	Muniyandi	49233	No	No	No	No	No	No	No	No	No	No	Yes	NC	Yes	Cloudy	20	Recovered
50	Veeramanickam	30967	No	No	No	No	No	No	No	No	No	No	Yes	NC	No	Cloudy	14	Recovered

Key: Sr Cr-Serum Creatinine; pO2& pCo2: partial pressures of oxygen and carbon dioxide respectively.

APACHE-II

S.no	Pt name	IP no	Age	Temp	MAP	Heart Rate	Resp rate	A-a PO2 / PaO2	Ar pH/H CO3	Sr Na mEq/ L	Sr K mEq/ L	Cr mg/dl	Hem atocrit %	WBC cells/ cu mm	GCS	CHP	A+B+C	% Mortality	Outcome
1	Subbaiyan	34507	2	1	0	0	0	0	0	0	0	0	0	1	0	0	4	5.10	Recovered
2	Rajendran	6225	0	1	0	2	0	0	0	0	0	2	1	1	0	0	7	7.60	Recovered
3	Tamilselvam	38979	0	0	0	2	0	0	0	0	0	0	0	1	0	0	3	4.40	Recovered
4	Veeranan	40630	6	1	0	2	1	0	0	0	0	0	0	1	0	5	16	23.50	Recovered
5	Chinna murugan	42041	0	3	2	2	1	3	2	2	1	6	2	1	3	0	29	67.20	Expired
6	Krishnan	7420	2	3	2	2	1	4	2	2	1	4	2	1	3	0	29	67.20	Expired
7	Ravi	36650	0	2	4	3	3	4	2	2	1	6	2	2	3	0	34	81.00	Expired
8	Arun Arockiaswamy	8170	0	0	0	2	0	0	0	0	0	0	0	0	0	0	2	3.80	Recovered
9	Ganesan	46294	2	4	2	2	3	2	3	0	1	2	1	2	3	0	27	60.50	Expired
10	Ganesh	39992	0	1	0	2	0	0	0	0	0	0	0	1	0	0	4	5.10	Recovered
11	Deivendran	43976	2	1	0	0	0	0	0	0	1	2	1	2	0	0	9	9.90	Recovered
12	Lingupandi	45458	3	3	2	2	1	2	2	1	1	3	1	2	2	5	30	70.30	Expired
13	Karthick	40141	0	1	0	2	0	0	0	0	0	0	1	2	0	0	6	6.70	Recovered
14	Marimuthu	41567	0	1	0	2	1	0	0	0	0	0	0	1	0	0	5	5.80	Recovered
15	Kannan	5448	0	0	0	2	0	0	0	0	0	0	0	1	0	0	3	4.40	Recovered
16	Rajendran	5447	2	1	0	2	0	0	0	0	0	0	0	2	0	5	10	11.30	Recovered
17	John Peter	31713	2	3	2	2	1	2	2	1	1	2	0	2	0	0	20	35.50	Recovered
18	Karthick Kumar	4269	1	1	0	2	1	0	0	0	0	0	2	0	0	0	7	7.60	Recovered
19	Mariappan	2810	3	1	0	2	1	0	0	0	0	0	0	0	0	5	12	14.60	Recovered
20	Meeranoideen	23149	3	3	0	2	1	2	2	0	0	0	1	1	0	0	15	21.00	Recovered
21	Valasuthurai	1790	3	3	0	2	1	2	2	0	0	0	1	2	5	5	21	38.90	Recovered
22	Arockiam	32669	3	3	2	3	1	2	2	2	1	4	2	2	2	5	33	78.60	Expired
23	Veluswamy	31057	0	1	0	2	0	0	0	0	0	2	0	0	0	0	5	5.80	Recovered
24	Anandamurugan	20630	0	3	2	2	1	2	2	0	1	3	0	1	0	0	17	26.20	Recovered
25	Periaswamy	33709	3	1	0	2	0	0	0	0	0	0	1	1	0	5	13	16.50	Recovered

26	Arumugam	23609	2	1	2	2	1	2	1	2	2	6	0	2	4	0	27	60.50	Expired
27	Kuzhanthairaj	23638	0	1	0	2	1	0	0	0	0	2	1	1	0	0	8	8.70	Recovered
28	Ilayaraja	6076	0	1	0	2	0	0	0	0	0	0	0	1	0	0	4	5.10	Recovered
29	Ponnasi	619	2	3	2	3	1	4	3	2	1	8	1	2	0	0	32	76.00	Expired
30	Ramachandran	87848	0	1	0	2	1	0	0	0	0	2	1	1	0	0	8	8.70	Recovered
31	Raja	85955	0	1	0	2	1	0	0	1	1	2	2	1	0	0	11	12.90	Recovered
32	Appadurai	75071	0	4	0	2	1	2	2	2	1	2	2	2	1	0	21	38.90	Recovered
33	Pappathy	44017	3	1	0	2	1	0	0	0	0	2	2	2	0	0	13	16.50	Recovered
34	Parameswari	36830	0	1	0	2	1	0	2	1	1	2	1	2	2	0	15	21.00	Recovered
35	Indrirakumari	21163	0	0	0	0	1	2	2	0	0	0	2	0	0	5	12	14.60	Recovered
36	Jothi	19780	2	3	0	2	1	2	2	0	0	4	1	1	0	0	18	29.10	Recovered
37	Chellammal	45582	5	1	2	2	1	4	2	2	1	6	2	1	3	0	32	76.00	Expired
38	Panchali	18391	3	1	2	2	1	4	2	1	3	4	2	2	3	0	30	70.30	Expired
39	Pappathy	37990	2	3	0	2	1	0	1	0	1	0	0	1	0	0	11	12.90	Recovered
40	Muthiah	24213	0	3	2	2	3	2	2	0	1	6	1	2	3	0	27	60.50	Expired
41	Subbiah	21553	0	3	2	2	3	2	2	0	0	6	1	2	4	0	27	60.50	Expired
42	Palaniswamy	18843	2	1	0	2	0	0	0	0	0	0	0	2	0	0	7	7.60	Recovered
43	Pandian	17343	2	1	0	2	0	0	0	0	0	0	0	1	0	0	6	6.70	Recovered
44	Karuppuswamy	32336	2	1	0	2	1	0	0	0	0	4	0	1	0	0	11	12.90	Recovered
45	Mallika	38761	0	2	0	2	1	0	0	0	0	0	1	0	0	0	6	6.70	Recovered
46	Nagarajan	20098	5	3	2	2	1	0	2	0	1	4	2	1	2	5	30	70.30	Expired
47	Jegan	10870	1	1	2	3	1	0	0	0	0	4	1	0	0	0	13	16.50	Recovered
48	Alagupandi	55255	2	3	0	2	1	2	0	2	1	4	1	2	0	0	18	35.50	Recovered
49	Muniyandi	49233	1	1	2	2	1	0	0	0	0	0	0	0	0	5	12	12.90	Recovered
50	Veeramanickam	30967	2	1	0	2	0	0	0	0	0	0	0	2	0	0	7	7.60	Recovered

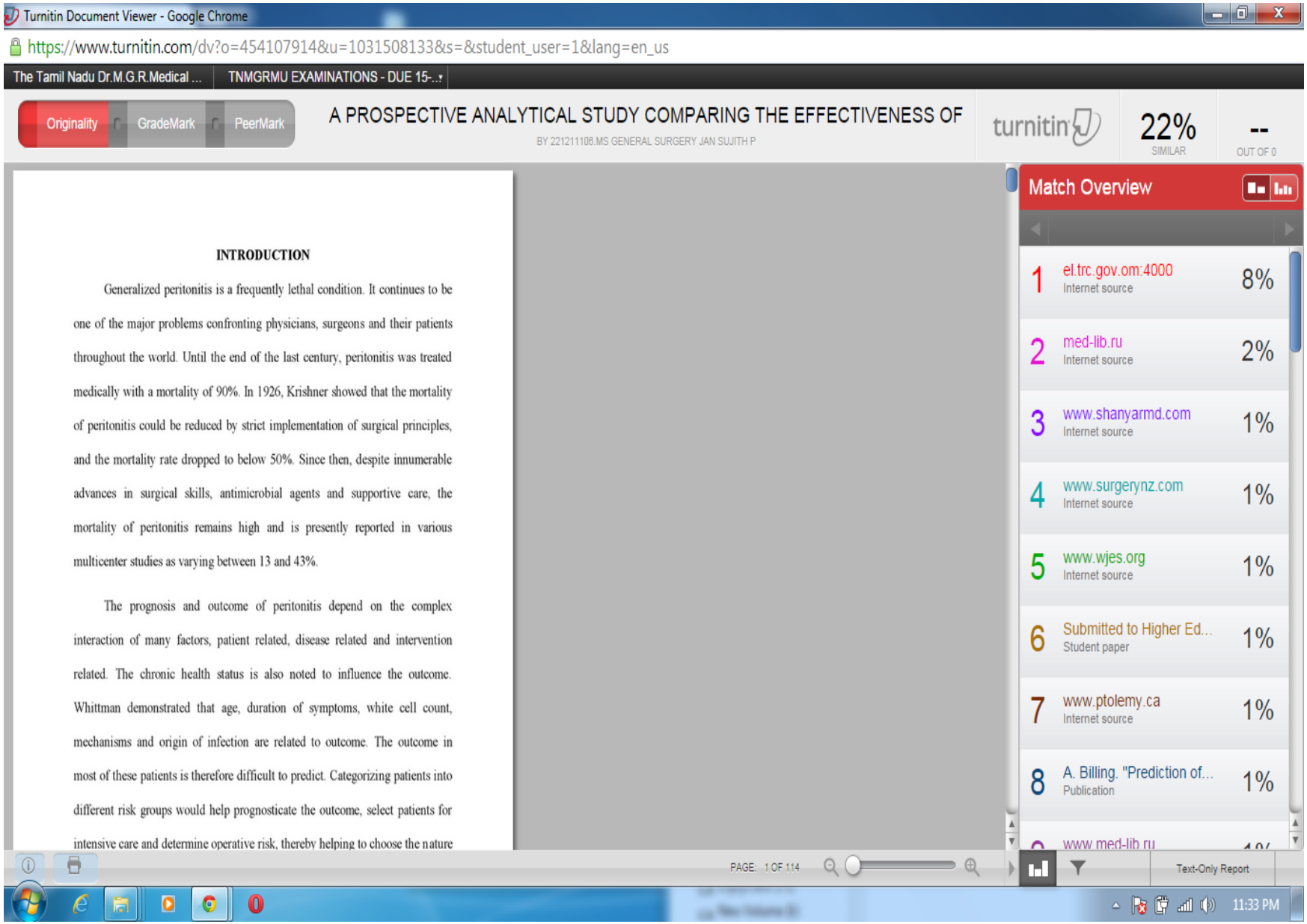
Key: MAP- Mean Arterial Pressure; (a-A)PO2- alveolar arterial gradient of oxygen; PaO2 Partial pressure of Oxygen; Sr Na/K+/HCO3 - Serum Sodium /Potassium/Bicarbonate respectively; Ar pH- arterial pH; GCS- Glassgow coma scale; CHP- Chronic health points WBC- White blood corpuscles. For numerical values please refer text.

p-POSSUM

S.no	Pt Name	IP no	Age	Cardiac sn	Resp H/O	ECG	SBP	PR	Hb	WBC count	Urea	Sr Na	Sr K	GCS	Physio Logic score	Operation severity	No of procedures	Operative blood loss	Malignancy	Peritoneal Soiling	Urgency	OP score	% Mortality	Outcome
1	Subbaiyan	34507	1	1	1	1	1	2	2	2	2	1	1	1	16	4	1	1	1	8	4	19	3.19	Recovered
2	Rajendran	6225	1	1	1	1	1	4	4	2	2	1	1	1	20	4	1	1	1	8	4	19	6.08	Recovered
3	Tamilselvam	38979	1	1	1	1	1	2	4	1	1	1	1	1	16	4	1	1	1	4	4	15	1.74	Recovered
4	Veeranan	40630	4	1	2	1	2	2	4	1	1	1	1	1	21	4	1	1	1	2	4	13	2.94	Recovered
5	Chinna murugan	42041	1	1	1	1	4	8	4	2	8	4	2	2	38	4	1	1	1	8	8	23	71.69	Expired
6	Krishnan	7420	1	1	1	8	4	8	4	2	4	4	2	2	41	4	1	1	1	8	8	23	80.80	Expired
7	Ravi	36650	1	1	1	1	8	8	8	1	4	4	2	2	41	4	4	1	2	8	8	27	88.66	Expired
8	Arun Arockiswamy	8170	1	1	1	1	1	2	1	1	1	1	1	1	13	4	4	1	1	8	8	26	5.54	Recovered
9	Ganesan	46294	1	1	2	1	4	4	8	2	2	1	2	2	30	4	4	2	4	8	8	30	65.95	Expired
10	Ganesh	39992	1	1	1	1	1	1	1	1	1	1	1	1	12	4	4	1	2	2	1	14	0.77	Recovered
11	Deivendran	43976	1	1	1	1	1	2	2	2	1	1	2	1	16	8	8	2	1	8	4	31	17.47	Recovered
12	Lingupandi	45458	1	1	2	2	4	8	4	4	4	2	2	2	36	4	4	2	1	8	8	27	77.05	Expired
13	Karthick	40141	1	1	1	1	1	2	2	2	1	1	1	1	15	2	1	1	1	2	4	11	0.80	Recovered
14	Marimuthu	41567	1	1	1	1	1	2	2	2	1	1	1	1	15	4	1	1	1	2	4	13	1.09	Recovered
15	Kannan	5448	1	1	4	1	1	4	4	2	1	1	1	1	22	4	1	1	1	2	4	13	3.46	Recovered
16	Rajendran	5447	1	1	1	1	2	4	2	2	1	1	1	1	18	4	4	2	1	2	4	17	3.28	Recovered
17	John Peter	31713	1	1	1	1	2	4	2	2	2	2	2	1	21	4	4	1	1	4	8	22	10.89	Recovered
18	Karthick Kumar	4269	1	1	1	1	1	2	2	1	1	1	1	1	14	4	4	4	1	4	4	21	3.10	Recovered
19	Mariappan	2810	1	2	2	1	1	4	2	1	1	1	1	1	18	4	4	2	1	2	8	21	5.93	Recovered
20	Meeramoideen	23149	1	1	1	1	1	4	2	2	1	1	1	1	17	4	4	1	1	8	4	22	5.85	Recovered
21	Valasuthurai	1790	1	1	1	1	2	8	2	2	2	1	1	1	23	4	4	1	1	8	4	22	14.64	Recovered
22	Arockiam	32669	1	1	4	1	4	4	4	2	4	2	2	2	31	4	4	2	2	8	8	28	62.71	Expired
23	Veluswamy	31057	1	1	1	1	1	4	4	2	2	1	1	2	21	4	4	1	1	8	4	22	10.89	Recovered

24	Anandamurugan	20630	1	1	1	1	3	4	1	2	4	1	2	1	23	4	4	1	1	8	4	22	14.63	Recovered
25	Periaswamy	33709	1	4	2	1	2	8	2	1	1	1	1	1	25	4	4	1	1	8	4	22	19.38	Recovered
26	Arumugam	23609	1	1	1	1	4	8	8	4	4	1	8	4	45	4	4	4	2	8	4	26	96.29	Expired
27	Kuzhanthairaj	23638	1	1	1	1	1	4	4	2	2	1	1	1	20	4	4	1	1	2	4	16	3.91	Recovered
28	Ilayaraja	6076	1	1	1	1	1	2	2	2	1	1	1	1	15	4	4	1	1	2	4	16	1.71	Recovered
29	Ponnasi	619	1	1	1	1	4	8	4	2	4	4	4	1	35	4	4	1	2	8	8	27	73.92	Expired
30	Ramachandran	87848	1	1	1	1	2	4	4	2	2	1	1	1	21	4	4	2	1	4	4	19	7.13	Recovered
31	Raja	85955	1	1	1	1	2	4	4	2	2	1	2	1	22	4	4	2	1	4	4	19	8.33	Recovered
32	Appadurai	75071	1	1	1	1	2	8	4	4	2	4	4	2	34	4	4	2	1	8	4	23	56.29	Recovered
33	Pappathy	44017	1	1	2	1	2	4	4	2	2	1	1	1	22	4	4	1	1	8	4	22	12.64	Recovered
34	Parameswari	36830	1	1	1	1	2	4	4	2	2	1	4	2	25	4	4	2	1	4	4	19	13.12	Recovered
35	Indirakumari	21163	1	1	1	1	2	4	4	1	2	1	1	1	20	4	4	1	1	4	4	18	5.26	Recovered
36	Jothi	19780	1	1	1	1	2	8	8	2	2	1	1	1	29	4	4	2	1	8	8	27	50.67	Recovered
37	Chellammal	45582	2	2	1	1	4	1	8	2	8	1	4	2	36	4	4	2	2	8	8	28	79.67	Expired
38	Panchali	18391	1	1	1	1	2	8	4	2	4	1	8	2	35	4	4	2	2	8	8	28	76.79	Expired
39	Pappathy	87990	1	1	1	1	1	4	2	2	1	1	2	1	18	4	4	1	1	2	4	16	2.82	Recovered
40	Muthiah	24213	1	1	1	1	8	8	4	4	8	1	2	2	41	4	1	1	1	8	8	23	80.80	Expired
41	Subbiah	21553	1	1	1	1	8	8	2	2	8	1	1	4	38	4	1	1	1	8	8	23	71.69	Expired
42	Palaniswamy	18843	1	1	1	1	1	2	2	2	1	1	1	1	15	4	4	1	1	4	4	18	2.32	Recovered
43	Pandian	17343	1	1	1	1	1	4	2	2	1	1	1	1	17	4	4	1	1	4	4	18	3.23	Recovered
44	Karuppuswamy	32336	1	1	1	1	1	4	4	2	2	1	1	1	20	4	4	4	1	2	4	19	6.08	Recovered
45	Mallika	38761	1	1	1	1	1	4	4	1	1	1	1	1	18	4	4	1	1	2	2	14	2.08	Recovered
46	Nagarajan	20098	4	2	4	1	4	4	4	2	2	1	4	1	33	4	8	2	4	2	4	24	77.79	Expired
47	Jegan	10870	1	1	1	1	8	8	4	1	2	1	1	1	29	4	4	2	1	2	4	17	20.52	Recovered
48	Alagupandi	55255	1	1	1	1	2	8	4	2	2	4	4	1	31	4	8	2	1	8	4	27	59.03	Recovered
49	Muniyandi	49233	1	8	1	1	2	4	4	1	1	1	1	1	26	4	4	1	1	8	4	22	22.17	Recovered
50	Veeramanickam	30967	1	1	1	1	1	2	2	2	1	1	1	1	15	4	4	1	1	4	4	18	2.32	Recovered

KEY: SBP-Systolic blood pressure; PR-Pulse rate; ECG- Electrocardiography; Hb-Haemoglobin; For numerical values please refer text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INTRODUCTION
Generalized peritonitis is a frequently lethal condition. It continues to be one of the major problems confronting physicians, surgeons and their patients throughout the world. Until the end of the last century, peritonitis was treated medically with a mortality of 90%. In 1926, Krishner showed that the mortality of peritonitis could be reduced by strict implementation of surgical principles, and the mortality rate dropped to below 50%. Since then, despite innumerable advances in surgical skills, antimicrobial agents and supportive care, the mortality of peritonitis remains high and is presently reported in various multicenter studies as varying between 13 and 43%.
The prognosis and outcome of peritonitis depend on the complex interaction of many factors, patient related, disease related and intervention related. The chronic health status is also noted to influence the outcome. Whittman demonstrated that age, duration of symptoms, white cell count, mechanisms and origin of infection are related to outcome. The outcome in most of these patients is therefore difficult to predict. Categorizing patients into different risk groups would help prognosticate the outcome, select patients for intensive care and determine operative risk, thereby helping to choose the nature
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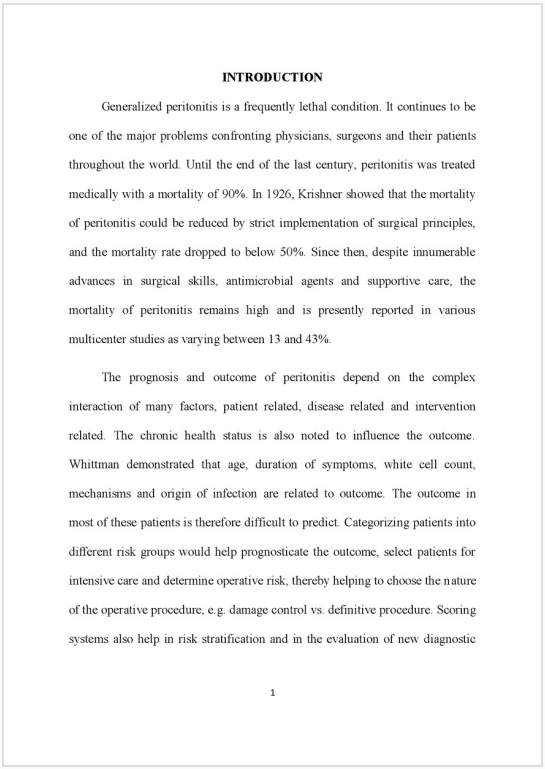

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